A STUDY OF THE ETIOLOGY, CONTRIBUTORY FACTORS AND CORRELATION OF THE CLINICAL AND LABORATORY PROFILES OF ANEMIA IN ELDERLY PATIENTS PRESENTING AT A TERTIARY HOSPITAL IN RURAL SOUTH INDIA

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

DOCTOR OF MEDICINE IN PATHOLOGY

(Branch III) M. D. (PATHOLOGY)

of

THE TAMIL NADU DR. M. G. R MEDICAL UNIVERSITY

CHENNAI- 600032

DEPARTMENT OF PATHOLOGY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 11

APRIL 2016

CERTIFICATE

I hereby certify that this dissertation entitled "**A STUDY OF THE ETIOLOGY, CONTRIBUTORY FACTORS AND CORRELATION OF THE CLINICAL AND LABORATORY PROFILES OF ANEMIA IN ELDERLY PATIENTS PRESENTING AT A TERTIARY HOSPITAL IN RURAL SOUTH INDIA"** is a record of work done by **Dr. G.JOSHILA NANDHINI** in the Department of Pathology, Tirunelveli Medical College, Tirunelveli, during her postgraduate degree course period from 2013- 2016. This work has not formed the basis for previous award of any degree.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF THE ETIOLOGY, CONTRIBUTORY FACTORS AND CORRELATION OF THE CLINICAL AND LABORATORY PROFILES OF ANEMIA IN ELDERLY PATIENTS PRESENTING AT A TERTIARY HOSPITAL IN RURAL SOUTH INDIA"** was done by me at Tirunelveli Medical College, Tirunelveli – 627011, during the period September 2013 to September 2015 under the guidance and supervision of **Prof. K. SHANTARAMAN, MD**, to be submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of MD DEGREE in PATHOLOGY BRANCH-III.

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PROTOCOL TITLE: A STUDY OF THE ETIOLOGY, CONTRIBUTORY FACTORS AND CORRELATION OF THE CLINICAL AND LABORATORY PROFILES OF ANEMIA IN ELDERLY PATIENTS PRESENTING AT A TERTIARY HOSPITAL IN RURAL SOUTH INDIA.

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Dear , Dr. G. Joshila Nandhini, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 27.12.2013.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

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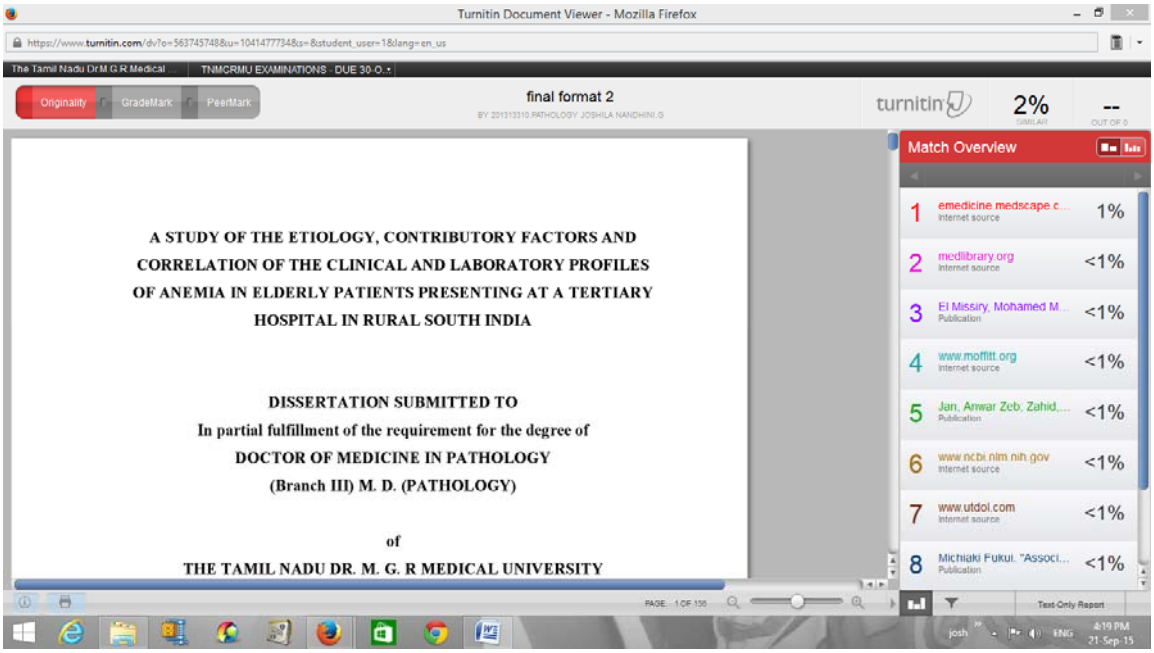

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Dr. JOSHILA NANDHINI G

ABBREVIATIONS

ACD	Anemia Of Chronic Disease
ADAMTS	A Disintegrin and metalloproteinase with thrombospondin motifs
AML	Acute Myeloid Leukemia
ATP	Adenosine TriPhosphate
AUE	Anemia Of Unexplained Etiology
A-CKD	Anemia of Chronic Kidney Disease
CKI	Chronic Kidney Injury
CLL	Chronic Lymphoid Leukemia
CML	Chronic Myeloid leukemia
CoA	Co enzyme A
DIC	Disseminated Intravascular Coagulation
DMT	Divalent Metal Transporter
dTMP	Thymidine mono phosphate
EPO	Erythropoietin
FAD	Flavin Adenine Diphosphate
GI	Gastro intestinal
GIT	Gastro Intestinal Tract
G6PD	Glucose 6 phosphate dehydrogenase
HCT	Hematocrit
HE	Hereditary Elliptocytosis
HgB	Hemoglobin
HS	Hereditary Spherocytosis
IDA	Iron Deficiency Anemia
IFN	Interferon
IL	Interleukin
MAHA	Micro Angiopathic Hemolytic Anemia
MCH	Mean Corpuscular Hemoglobin
MCHC	Mean Corpuscular Hemoglobin Concentration
MCV	Mean Corpuscular Volume

MDS	MyeloDysplastic Syndrome
NAD	Nicotinamide adenine Diphosphate
NCNC	Normocytic Normochromic
NHANES	United Nations national Health And Nutritional Examination Survey
Pg	Picograms
RBC	Red Blood Cell
RDW	Red cell Distribution Width
RNA	Ribonucleic acid
sTFR	Soluble Transferrin Receptor
TGF	Tissue Growth Factor
TNF	Tissue Necrosis Factor
TTP	Thrombotic Thrombocytopenic Purpura
vWF	Von Willebrand Factor
WBC	White Blood Cell
WHO	World Health Organisation

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ABSTRACT

Background: Anemia is defined as a state of decreased oxygen carrying capacity of the blood wherein the hemoglobin is less than 13 g/ml in men and less than12 g/ml in non pregnant women by WHO reference standards. The demographic transition with ageing of population is a global phenomenon and in recent years there has been an increasing international awareness of health issues relating to aging population. The prevalence of anemia among elderly Indians, as reported in Indian cross sectional studies is between 6 and 30 % among men and between 10 and 20 % among women. Thus anemia represents an emerging global health problem producing a negative impact in the quality of life among the elderly and requiring greater allocation of health resources.

Aims: To identify elderly patients with anemia and study the etiology, clinical parameters, laboratory parameters in these patients.

Methods: The cases received in the hematology department for peripheral smear reporting were screened and 100 cases that showed hemoglobin levels lower than standard WHO values were chosen. The entire medical history was obtained from each of the cases including previous medical reports and imaging studies. Blood samples were collected from all patients in plain vacutainers and EDTA containing vacutainers. Complete blood counts for all the samples was done using SYSMEX 3 part differential analyzer and the RBC indices was noted along with total WBC count, differential count, platelet count and hematocrit. ESR for all cases was done using Westergren tube. Peripheral smears were made for all the samples and stained using Leishman stain. Supravital staining using methylene blue was done for reticulocyte counting. Ferritin, iron and TIBC for all samples was done using SPINREACT kits for all the three and values obtained using colorimetric methods. The results obtained by the above tests was analyzed and the different etiologies, clinical features, intensity of anemia, peripheral smear picture and iron parameters were studied and compared with other studies.

Results: Among the etiologies, iron deficiency anemia was seen in 43% of patients, anemia of chronic disease in 32%, anemia of chronic kidney disease and unexplained anemia in 11 % each and 1 case β thalassemia. Among clinical features the commonest symptom was easy fatiguability and commonest sign was pallor. Peripheral smear study showed that normocytic normochromic anemia was commonest type.

Conclusion: This study showed that the commonest cause for anemia among elderly patients is iron deficiency anemia followed by anemia due to chronic disease and also that it can be asymptomatic which is incidentally stumbled upon when one is evaluated for other symptoms. Not many clinical signs are consistent with anemia except for pallor even which can be absent in cases of mild anemia. Even though iron deficiency anemia is the commonest cause the peripheral smear studies in this study showed that normocytic normochromic picture was the commonest even when MCV levels were suggestive of microcytic anemia. Geriatric anemia is a disease that often goes unreported hence every effort should be made to identify the disease and evaluate the cause and it should not be ignored as merely being a part of ageing, for the consequences of anemia can have higher morbidity in the elderly.

Key Words: Geriatric anemia, Anemia in elderly, Iron deficiency, Iron parameters, Peripheral smear.

INTRODUCTION

Anemia is defined as a state of decreased oxygen carrying capacity of the blood wherein the hemoglobin is less than 13 g/ml in men and less than12 g/ml in non pregnant women by WHO reference standards [1].The demographic transition with ageing of population is a global phenomenon and in recent years there has been an increasing international awareness of health issues relating to aging population[5,6]. According to a 2004 report of the United States National Health and Nutritional Examination Survey (NHANES) III, 10 % of Americans 65 years and older are anemic, rising to a 25 % of men and 20 % of women 85 years and older [3].The prevalence of anemia among elderly Indians , as reported in Indian cross sectional studies is between 6 and 30 % among men and between 10 and 20 % among women [4].Thus anemia represents an emerging global health problem producing a negative impact in the quality of life among the elderly and requiring greater allocation of health resources[7].

AIMS OF THE STUDY

1. To identify elderly patients (> 65 years of age) with anemia based on WHO criteria,
2. To study clinical features, etiology in those diagnosed with anemia,
3. To analyse the laboratory parameters in these patients.

REVIEW OF LITERATURE

Geriatric anemia is a unique clinical entity for several reasons. Firstly, its diagnosis poses a challenge because several features of anemia are easy to overlook [8]. The onset of symptoms and signs is usually insidious and many elderly patients adjust their activities as their bodies make physiological adaptations [8]. Secondly, in contrast to younger ages anemia is more common in elderly men than women with a high incidence occurring in men older than 85 years[8].Thirdly, the etiology of geriatric anemia varies considerably from anemia in younger age [8].

ETIOLOGY:

Anemia in the elderly is multifactorial with the most common causes being nutritional deficiencies including iron, B 12, folate deficiencies, anemia of chronic inflammation/ disease and chronic kidney disease [9, 10] and other less common causes being diseases of the marrow, hemolytic anemias and endocrinopathy. Most of the time despite a complete evaluation a significant number of cases have no etiology identified [9, 10].

TABLE NO. 1. CAUSES OF ANEMIA IN ELDERLY:[11]

Cause	**Prevalence**
Iron deficiency	15 to 30 %
Chronic disease/inflammation	30 to 45 %
Chronic kidney disease	8%
Post hemorrhagic	5 to 10 %
Vitamin B12 or folate deficiencies	5 to 10 %
Myelodysplastic syndromes	5%
Chronic leukemia or lymphoma	5%
Unexplained	14% to 45%

Data from Joosten E, Pelemans W, Hiele M, et al. Prevalence and causes of anaemia in a geriatric hospitalized population. *Gerontology*. 1992; 38(1-2):111-7.

NUTRIENT DEFICIENT ANEMIAS:

Nutrient deficient anemias are significant cause of anemia in the elderly. Iron, folate or vitamin B-12 deficiencies maybe seen, but mostly a combination of the three occurs [9].

ANEMIA DUE TO IRON DEFICIENCY

IRON METABOLISM

Iron is derived from diet either as heme iron contained in animal products or as non-heme iron in plant products like cereals, pulses and vegetables. Heme iron is absorbed better when compared to non heme iron. Iron in the body can be divided into storage and functional compartments. About 80% of the iron is in functional form as in hemoglobin, myoglobin and certain enzymes such as catalase and cytochromes, the remaining iron is stored in the body in the form of ferritin and hemosiderin, which is a permanent non reusable form.

Iron balance

Dietary iron is absorbed in proximal duodenum. Luminal non heme iron is mostly in ferric state and must first be reduced to ferrous form by ferrireductases like b cytochromes[15]. Ferrous iron is then transferred across apical membrane by divalent metal transporter 1 (DMT 1). Heme iron is directly transported into enterocyte by a heme transporter. Iron can now be either circulated or stored. Iron that is destined for circulation is transported across the basolateral membrane by ferriportin, a process that is coupled to oxidation of ferrous iron to ferric form by hephastin and ceruloplasmin. Newly absorbed iron binds to transferrin and is rapidly delivered to erythroid progenitors.

Role of transferrin

Transferrin is a glycoprotein with a molecular weight of approximately 80 kD which has iron binding capacity, is synthesized by the liver and enables delivery of iron to cells including erythroid precursors[16]. The eythroid precursors have high affinity receptors for transferrin which mediate iron absorption through endocytosis[15].

Role of ferritin

Ferritin is a ubiquitous protein that is found in highest levels in the liver, bone marrow and spleen[16]and serves to store iron as reusable, non toxic molecule while free iron is ionized and hence highly toxic. Apoferritin, is an apoprotein present in the intestinal mucosa, that binds to free ferrous iron forms ferritin[17] and stores it in ferric state. Intracellular ferritin is located in cytosol and in lysosomes in which partially degraded protein shells of ferritin accumulate into hemosiderin granules. Under steady state conditions serum ferritin levels correlate with body iron stores so is the most effective way to diagnose anemia and when levels are below 16 ng/ml iron deficiency is highly likely [14].

Role of hepcidin

It is a small circulating peptide that is synthesized and released from the liver in response to increases in intrahepatic iron levels and inhibits iron transfer from enterocyte and macrophages to plasma by binding to ferriportin causing it to be endocytosed and degraded[16]. As hepcidin levels increase, iron becomes trapped within duodenal cells in the form of mucosal ferritin and is lost as these cells get sloughed.

IRON DEFICIENCY

An estimated 16 % of anemic elderly have iron deficiency [9] which occurs when the rate of iron utilization exceeds the rate of intestinal absorption and may be caused by

1. inadequate intake
2. impaired absorption
3. increased requirement
4. chronic blood loss

Inadequate intake

In the developing countries iron is mainly obtained in the non heme form from cereals, pulses, vegetables and fruits which is a poorly absorbed form of iron[16] in contrast to the Western world where most of the iron is in the heme form from animal products. Hence people in developing countries owing to their diet habits are at risk for iron deficiency where elderly are at an even further risk due to decreased intake of food and poor dentition[16].

Impaired absorption

Iron absorption is defective in cases of celiac sprue, fat malabsorption syndromes, diarrhea, following gastrectomy etc[16]. Commonly used medications for acid peptic disease like antacids, proton pump inhibitors, H_2 blockers also inhibit iron absorption by decreasing acid content in stomach.

Chronic blood loss

Iron deficiency in the elderly always leads to an evaluation of the gastrointestinal (GI) tract as a possible source of bleeding. 20 to 40 % of patients

have upper GI bleed from peptic ulcer disease, gastritis, esophagitis or gastric cancer [12], with 15 to 20 % cases having loss from the colon due to causes like angiodysplasia, polyps, colitis [12].

ANEMIA DUE TO VITAMIN B-12 DEFICIENCY

Vitamin B -12 is a complex organo-metallic compound called cobalamin essential for DNA synthesis, maturation and normal functioning of the nervous system. Micro-organisms are the ultimate source of this vitamin, vegetarian diets are deficient in this vitamin. Vitamin B 12 acts as cofactor in conversion of homocysteine to methionine, a reaction which yields tetrahydrofolate through several steps. Tetrahydrofolate is essential since it is required for conversion of deoxyuridine phosphate to deoxythymidine monophosphate, an immediate precursor of DNA. Deficiency of vitamin B 12 causes reduced availability of tetrahydrofolate leading to impaired DNA synthesis. Anemia due to vitamin B 12 and folate is termed megaloblastic anemia and is found to have an incidence of 14 % according to NHANES III study [18].

PATHOGENESIS:

ABSORPTION OF VITAMIN B-12

This is done by the presence of a factor called the intrinsic factor which is secreted by the parietal cells of gastric fundic mucosa.B-12 from the diet gets bound to R- Binders, also called transcobalamin I or cobalophilin, in saliva. When it reaches the duodenum it gets dissociated from the R-binder and gets bound to intrinsic factor and remains bound till it reaches the ileum. In the ileum, it is endocytosed by ileal enterocytes and gets associated with a carrier protein

transcobalamin II and is secreted into the plasma which delivers B – 12 to the required sites rapidly.

PERNICIOUS ANEMIA

It is a disease of older adults uncommon in ages below 30 years with a suspected genetic predisposition and a prevalence of 0.1% in the general population and 1.9% in subjects over the age of 60 years[19]. It is due to autoimmune attack on the gastric mucosa and there are three types of autoantibodies are present[16]:

Type I which blocks the binding of B-12 to intrinsic factor and is the cause in 75% of the patients, type II prevents binding of IF-B12 complex to ileal receptor and type III which acts against α and β subunits of gastric proton pump and is found in 90 % of persons not specific for pernicious anemia. The autoantibodies are of diagnostic utility, they are not the primary cause of gastric pathology. The autoreactive T-cell response initiates gastric mucosal injury and triggers formation of autoantibodies which further exacerbate epithelial injury and cause the mass of intrinsic factor secreting cells to fall below a threshold, when anemia develops. Pernicious anemia is found to be associated with other autoimmune diseases such as Hashimotos thyroiditis, type 1 diabetes mellitus [19].

OTHER CAUSES OF B-12 DEFICIENCY [16]

1. Achlorhydria and loss of pepsin secretion.
2. Following gastrectomy IF is unavailable
3. Loss of exocrine pancreatic function, B-12 cannot be released from R-binder complex.

4. Ileal resection can remove or damage the site of IF-B12 absorption
5. Tapeworm infestation when present compete with the host for B 12 and induce deficiency
6. In conditions like hyperthyroidism, disseminated cancer, chronic infections an increased demand for B12 can produce a relative deficiency.

The vitamin B-12 levels have been used to estimate the prevalence of B-12 deficiencies with a threshold of 200 pg/ml or 148pmol/L[20]. Unpublished data from the National Health and Nutrition Examination Survey (NHANES) 2001-2004 stratified by age have estimated that 1 (3.2%) of every 31 adults 51 years of age or older in the United States will have a low vitamin B_{12} serum level (≤ 200 pg/mL) and The Framingham study with a cohort of non institutionalized adults 67 through 96 years of age found that 5.3% of the participants had serum vitamin B12 levels below 200 pg/mL[20].

ANEMIA DUE TO FOLATE DEFICIENCY

Anemia due to folate deficiency alone was found to constitute 6 % of all cases and with vitamin B-12 deficiency they constituted 2 % of all cases[9].Folic acid is obtained primarily form plant foods and is essential for the synthesis and repair of DNA, as well as to act as a cofactor in a number of biological reactions. All of the functions of folic acid is performed by tetrahydrofolate(FH_4) and other derivatives where the FH_4 derivatives act as intermediates in transfer of one carbon units such as formyl and methyl groups to various compounds[16]in reactions such as (i) purine synthesis, (ii) conversion of homocysteine to

methionine (iii) deoxythymidylate synthesis,of which dTMP is perhaps the most important biologically since it is important for DNA synthesis.

Sources of folate are plant foods like lettuce, spinach, asparagus and broccoli where it is in the form of folylpolyglutamates and get easily destroyed by cooking. The intestinal conjugases split the polyglutamates into monoglutamates that are readily absorbed into proximal jejunum where they are modified into 5-methyl tetrahydrofolate, the normal transport form of folate[16].

The causes for folate deficiency in elderly include .

1. Decreased intake or malabsorption
2. Hyperutilisation of folic acid by metabolic processes caused by malignancy, Crohn's disease, rheumatoid arthritis
3. Certain drugs that act as folic acid antagonists like methotrexate,(used for a variety of conditions in the elderly like rheumatoid arthritis, lupus, psoriasis, asthma) phenytoin, trimethoprim, chemotherapeutic drugs damage DNA or inhibit DNA synthesis through several mechanisms and cause megaloblastic changes.
4. The patients on dialysis for renal failure tend to lose folic acid in the dialysis fluid[13].

ANEMIA OF CHRONIC INFLAMMATION:

It was formerly called anemia of chronic disease and it is a hypoproliferative anemia defined as having low serum iron and RBC levels despite normal levels iron stores occuring in the setting of chronic inflammation and is associated with decreased serum iron, decreased total iron binding capacity

, increased serum ferritin levels and abundant stored iron in macrophages. The illnesses associated with this type of anemia include chronic microbial infections, chronic immune disorders and neoplasms. It is the most common cause of anemia in the elderly constituting 32 % of all etiologies according to NHANES III study [12].

Older adults tend to have higher levels of pro-inflammatory cytokines secondary to multiple co-morbid illnesses like atherosclerosis, diabetes mellitus and malignancies. The cytokines implicated in anemia of chronic inflammation include TNF α[21,22], IL 1 [23,24], IFN α[25], IL-6[24]. The pathophysiology implicated in anemia of chronic inflammation has a number of mechanisms, the most important of which is through hepcidin. Hepcidin as discussed earlier serves as a primary regulator of iron homeostasis. It serves to inhibit ferroportin which transports iron out of storage units. IL- 6 is found to increase hepcidin expression [26, 27], trapping iron within the macrophages and starving the erythroid precursors of the iron. In addition these progenitors do not proliferate adequately because erythropoietin levels are inappropriately low for the degree of anemia due to the action of IL-1, TNF α and TGFβ. TNF also inhibits the erythroid colony forming units thereby reducing erythropoeisis. Also cytokine IL-6 causes increased plasma volume thereby leading to a dilutional anemia. The erythrocyte survival is shortened in the setting of chronic inflammation which has been attributed to an extra corpuscular hemolysis. The cause for iron sequestration in setting of chronic inflammation is considered to be the body's defense in fending off certain bacteria

dependant on iron for survival. The anemia is usually mild with either normocytic normochromic or microcytic hypochromic RBCs.

RENAL INSUFFICIENCY:

Chronic kidney disease is an important cause of anemia in elderly due to the fact that renal function declines with age[26,27] which is further decreased by diseases like diabetes mellitus and hypertension. Anemia in chronic renal disease is attributed mainly to decreased erythropoietin and reduced RBC survival.

PATHOGENESIS:

Role of Erythropoietin *(EPO)*

It is a glycoprotein which is a hematopoietic growth factor which serves primarily to regulate RBC production. Secretion of EPO mainly occurs in the interstitial fibroblasts in the kidney in close association with peritubular capillary and proximal convoluted tubule with a lesser contribution from perisinusoidal cells in the liver[28]. Reduced tissue oxygenation potently induces a logarithmic increase in the EPO synthesis. EPO enhances erythrocyte production mainly by inhibiting apoptosis of erythroid progenitor cells and to a lesser degree by enhancing erythroid progenitor proliferation and differentiation. Serum erythropoietin levels have been shown to be inappropriately low at a creatinine clearance of < 40 %[29,30]. Mild decreases in hemoglobin may be detected at a creatinine clearance of 40- 60 ml/min[31,32]. Anemia is considered to be an indication of end stage kidney disease but women and diabetics may develop this complication at an earlier stage of the disease [33].

Reduced red cell survival in kidney disease

Some studies have shown that plasma from uremic patients can depress heme synthesis [34] or inhibit growth of erythroid colonies [35, 36]. Inhibitors of multipotential stem cell have also been found [37]. Secondary hyperparathyroidism in renal failure patients may also be a reason for marrow suppression [38]. Survival of RBC is found to be decreased and it has been discovered that the survival is related to the level of azotemia [39].

ANEMIA DUE TO ENDOCRINOPATHIES:

1. ***Variations in thyroxine levels***

The prevalence of anemia in hypothyroid patients is 20 – 60 % [43]. Thyroid hormones are necessary for hemoglobin synthesis in adults and maturation of hemoglobin in the fetus [40, 41] hence hypothyroidism results in anemia[42]. The anemia in hypothyroidism is commonly normocytic[44] but occasionally macrocytic [45]. Both hypothyroidism and hyperthyroidism have been known to cause pernicious anemia[46].

2. Androgen deficiency

Testosterone decreases with aging in men[47] and the greater rate of fall hemoglobin levels in men than in women suggests that falling testosterone maybe one of the causes of unexplained anemia. Testosterone accentuates proliferation of erythroid burst forming units and colony forming units by stimulating specific nuclear receptors and this effect is abolished by pretreatment of marrow cells with cyperoterone flutamide which block binding of androgens to their nuclear receptors [48]. The erythropoietic effects of testosterone is best explained by the

fact that following orchidectomy or androgen deprivation therapy for prostatic carcinoma hemoglobin levels fall by 1.2 g/dl to 1.5 g/dl on an average[49,50].

Hypopituitarism

Anemia due to hypopituitarism is usually normocytic normochromic [51] and results chiefly from deficiency of hormones of the target organs controlled by the pituitary [51]. Panhypopituitarism produces effects on RBC production by decreasing oxygen consumption [52] which in turn decreases erythropoietin secretion and red cell mass until new equilibrium is established between oxygen supply and demand [51].

ANEMIA DUE TO MYELODYSPLASTIC SYNDROMES (MDS):

Myelodysplastic syndromes represent a heterogeneous group of disorders characterized by clonal hematopoiesis and peripheral blood cytopenias resulting from bone marrow dysfunction [53]. Myelodysplasia is more common in elderly patients, more than 75 % of patients are over 60 years of age [53] with recent estimates of approximately 45000 new cases per year .Anemia in conjunction with macrocytosis, thrombocytosis or neutropenia in the absence of other causes should raise the suspicion of myelodysplasia[54,55]. The prevalence of anemia due to MDS has been estimated between 9 and 16 % based on three studies, NHANES III, Chicago and Stanford hospital and Clinics, VA Palo Alto Health Care system [9,63,64].

PATHOGENESIS:

The basic pathophysiology in MDS involves increased apoptosis and proliferation. The proliferation is due to clonal expansion of a multipotent

hematopoietic progenitor that is capable of granulocyte, monocyte, erythrocyte and megakaryocyte differentiation[56, 57, 58, 59]. Also increased levels of apoptotic mediators are present including TNF – α , FAS antigen (CD -95) and calcium dependant kinase [60, 61, 62]. The proliferation of progenitor cells results in a hypercellular marrow but failure to accumulate adequate number of mature cells results in cytopenia.The peripheral smear picture is that of macrocytosis or dimorphic RBC picture[65].

ANEMIA DUE TO PRIMARY HEMATOLOGIC DISORDERS:

A variety of other primary hematologic disorders have anemia as a manifestation. Anemia in the elderly can be due to primary hematological malignancies like AML, CLL, aplastic anemia or Multiple myeloma.

ACUTE MYELOGENEOUS LEUKEMIA

In normal hematopoiesis, the myeloblast is an immature precursor of myeloid white blood cells; a normal myeloblast will gradually mature into a mature white blood cell. In AML, though, a single myeloblast accumulates genetic changes which "freeze" the cell in its immature state and prevent differentiation.[66]Such a mutation alone does not cause leukemia; however, when such a "differentiation arrest" is combined with other mutations which disrupt genes controlling proliferation, the result is the uncontrolled growth of an immature clone of cells, leading to the clinical entity of AML.[67]. Anemia due to AML result from the growth of leukemic clone cells, which tends to displace or interfere with the development of normal blood cells in the bone marrow[68].

CHRONIC LYMPHOID LEUKEMIA

Chronic lymphoid leukemia (CLL), is the most common type of leukemia in adults[69] affecting 2 to 6 individuals per 100□000 per year. It is a neoplastic disease characterized by the accumulation of small mature-appearing CD5+ B lymphocytes in the blood, marrow, and lymphoid tissues. The causes of this disease are unknown, although genetic factors likely contribute to its development. Anemia develops due to crowding of bone marrow with proliferating lymphocytes thus decreasing erythroid precursors.

MULTIPLE MYELOMA

Multiple myeloma (MM) is a malignant plasma cell disorder that accounts for approximately 10% of all hematologic cancers[70]. The disease is characterized by monoclonal proliferation of plasma cells together with overproduction of a monoclonal antibody,[71] often accompanied by anemia, hypercalcemia, renal insufficiency, or bone lesions[72]. Approximately 97% of MM patients develop anemia during the course of their illness, and 70% are anemic at diagnosis. The anemia is usually normocytic normochromic,[73] serum-iron levels are normal to low, serum ferritin is high, and hemosiderin is prominent in bone marrow macrophages.[74] This suggests than iron release from reticulo endothelial macrophages is impaired, consistent with anemia of inflammation[75].

APLASTIC ANEMIA

It is a hypoproliferative state which can occur due to several causes some of which are toxins like benzene, drugs like antimetabolites, anthracyclines, autoimmune diseases and inherited causes like Fanconi's anemia and Diamond

Schwachman syndrome[79,80,81].According to the International Aplastic Anemia and Agranulocytosis Study the overall incidence of aplastic anemia is 2 cases/ I million with a higher incidence in southeast asia[76]. It commonly presents in the 2nd decade with a smaller peak in incidence after the age of 60 years [77, 78]. Immune injury to the marrow after drug-, viral-, or toxin-induced marrow aplasia could result from induction of neoantigens that provoke a secondary T cell–mediated attack on hematopoietic cells [82] causing reduced hematopoietic precursors leading to the pancytopenia.

ANEMIA SECONDARY TO BONE MARROW INFILTRATES – MYELOPHTHISIC ANEMIA:

This is a type of anemia that results from marrow infiltration by tumour cells or non hematopoietic tissue. The marrow environment is susceptible to infiltration by almost all cancers but the most common are lung, breast and prostate cancers [83]. The anemia is usually mild to moderate and peripheral smear may show presence of tear drop RBCs and nucleated RBCs a feature suggestive of marrow infiltration. Bone marrow biopsy is the most reliable procedure to confirm the diagnosis and should performed in all patients suspected of having myelophthisic anemia [83, 84, 85].

ANEMIA DUE TO HEMOLYTIC DISEASE:

Hemolytic anemias are the result of RBC deficiency due to premature destruction of RBC either within blood vessels (intravascular) or elsewhere (extravascular) resulting in shortened RBC lifespan and the marrow is unable to

compensate for the loss resulting in anemia .They can be classified as inherited and acquired. Inherited causes for hemolysis include,

INTRINSIC MEMBRANE DEFECT:

1. Hereditary spherocytosis, hereditary elliptocytosis,:

In hereditary spherocytosis there is defect in proteins of erythrocyte membrane including ankyrin, band 3, β spectrin, α- spectrin and protein 4.2. This leads to increased membrane fragility leading to loss of membrane surface area relative to intracellular volume, accounting for the spheroidal shape and decreased deformability of red cell[86]. The size of fenestrations in the venous sinuses in the spleen is small relative to the RBC size, and to pass through requires significant deformability of the red cell and its membrane. This, however, is a major problem for spherocytes, which have lost surface area and are dehydrated and get lysed as they pass through. It presents with chronically compensated mild to moderate hemolytic anemia and its diagnosis is based on presence of spherocytes in peripheral smear, a positive family history and a negative direct antiglobin test. The mean corpuscular hemoglobin concentration is frequently elevated [87].

Hereditary elliptocytosis (HE) and variants

Similar to HS, these group of disorders occur due to defects in RBC membrane proteins of which spectrin defect is commonest. The mutation impairs ability of spectrin dimers to self associate into dimers and tetramers thereby disrupting the membrane cytoskeleton and reducing the deformability of RBC s as they pass through splenic sinusoids.

Hereditary pyropoikilocytosis is a variant of HE and a severe congenital hemolytic anemia that presents with RBC fragments, poikilocytes and microspherocytes on peripheral smear.

2.Hemolysis due to abnormal hemoglobins

This can be due to either reduced synthesis of globin chains (quantitative) or defective synthesis of globin chains(qualitative).

QUANTITATIVE DEFECTS

Thalassemia

This is a group of congenital anemia in which there is deficient synthesis of one or more globin subunits of normal hemoglobin. There is either production of either α or β (called α thalassemia or β thalassemia respectively) chains as a result of which there is accumulation of free globin chains that cause lysis of RBC precursors. It can occur in three forms thalassemia major in which there is severe anemia requiring frequent transfusions, thalassemia minor or thalassemia intermedia in both cases there is mild to moderate anemia. The prevalence of anemia due to thalassemia in elderly ranges from 12 – 20 %[9, 63, 64].

QUALITATIVE DEFECTS

Sickle Cell Anemia

Sickle hemoglobin is a mutant hemoglobin in which valine has been substituted for the glutamic acid normally at the sixth amino acid of the β- globin chain. This hemoglobin becomes polymerized and becomes poorly soluble when the oxygen tension is lowered and red cells that contain this hemoglobin become distorted and rigid. Hemolysis in sickle cell anemia results from the lysis of

complement-sensitive red cells [88] and Hb lost during sickling or shear-induced membrane fragmentation[89]. Extravascular hemolysis may occur by two mechanisms: monocyte and macrophage recognition and phagocytosis of red cells that have undergone sickling- or oxidation-induced membrane[90] and physical entrapment of rheologically compromised red cells[91] Sickling and oxidation-induced membrane changes promote cell dehydration and clustering of membrane protein band 3 which leads to accumulation of IgG and complement on the sickle cell surface [92].

3. Erythrocyte enzymopathies:

Glucose-6-phosphate dehydrogenase (G6PD) is a highly conserved housekeeping enzyme and rate-limiting enzyme of the pentose phosphate pathway in all cells[93].The pentose phosphate pathway (PPP) converts glucose to ribose-5-phosphate, a precursor to RNA, DNA, ATP, CoA, NAD, and FAD and also provides reductive potential in the form of NADPH[94]. G6PD is a ubiquitous enzyme that must be quite ancient in evolution because it has been found in all organisms, from prokaryotes to yeasts, to protozoa, to plants, and animals [95,96]. G6PD deficiency results from mutations[97] in the G6PD gene and is well-known common cause of hemolytic anemia in human. RBCs lack other pathways to generate NADPH so G6PD deficiency becomes especially lethal in red blood cells, where any oxidative stress will result in hemolytic anemia.

Acquired Causes Of Hemolytic Anemia

Acquired causes of hemolysis include

1. Hemolysis due to mechanical injury to RBCs

2. Immune mediated injury to RBCs

3. Drug induced injury to RBCs

4. Hemolysis due to infections

Mechanical injury to RBCs can occur due to several causes

(i) Cardiac hemolysis

(ii) Microangiopathic haemolytic anemia

Cardiac hemolysis

This is due to injury to the red blood cells owing to turbulence in the heart chamber because of the pressure gradient created most commonly by prosthetic valves. It can also be caused due to aortic stenosis, mitral valve disease, coarctation of aorta.

Microangiopathic hemolytic anemia (MAHA)

This encompasses several etiologies, the common mechanism in these disorders is a microvascular lesion that results in luminal narrowing often due to deposition of fibrin and platelets. The causes of MAHA are

a. Thrombotic thrombocytopenic purpura (TTP)

b. Disseminated malignancies

TTP:

In this disease there is deficiency of the enzyme ADAMTS13 also designated as Von Willebrand Factor metalloprotease whose function of ADAMTS13 is to degrade the high molecular weight multimers of Von Willebrand Factor (vWF). In its absence the vWf multimers accumulate in plasma and promote platelet activation and aggregation. The deficiency can be either

acquired or inherited where in the acquired form there is antibody against the ADAMTS13 and in the inherited form there is deficiency of ADAMTS13.

DISSEMINATED MALIGNANCIES

Hemolysis due to disseminated malignancies is common in pancreatic, lung and prostatic carcinomas [101, 102]. Hemolysis can occur either due to DIC or intravascular tumour emboli. In case of DIC, a protease found in mucin secreted by adenocarcinoma directly activates factor X. Subsequently the coagulation cascade gets activated resulting in deposition of fibrin, formation of intravascular hyaline thrombi which cause shearing of red cells. Intravascular tumor emboli disrupt the endothelium, promoting deposition of fibrin, adherence of platelets, intimal hyperplasia, and vascular hypertrophy[98,99,100].Peripheral smear shows presence of moderate to severe anemia, schistocytes, helmet cells, microspherocytes, polychromasia and nRBCs[100], normal to high WBC count with a left shifted differential count[99, 100, 103]. Marrow aspiration and biopsy reveal erythroidhyperplasia , normal to high numbers of megakaryocytes and tumour invasion in 55 % of patients[103].

IMMUNE MEDIATED HEMOLYSIS

It is characterized by shortened RBC lifespan and presence of antibodies directed against the red cells. The antibodies can either be warm antibody type or cold antibody type.

Warm antibody type:

This type is predominantly due to IgG type immunoglobulin which acts best at a temperature of 37^0 C with or without complement proteins. The

autoantibody coated RBCs are trapped by macrophages in Billroth cords of spleen to a lesser extent by Kupffer cells in liver.

Cold antibody type:

It is caused by IgM antibodies that bind red cells at low temperatures (0^0C – 4^0C). The IgM antibodies bind to red cells causing agglutination and rapid complement fixation. This deposits sublytic quantities of C3b which leads to removal of affected red cells.

DRUG MEDIATED HEMOLYSIS:

Injury to RBCs due to drugs can occur due to several mechanisms:

(i) Drug adsorption mechanism:

Drugs like penicillin binds firmly to proteins on RBC membrane which is recognized by IgG antibodies and these RBCs are sequestered in the spleen.

(i) Ternary complex mechanism

In drugs like quinidine the antibodies recognize a complex of the drug and a membrane protein, fix complement and cause intravascular hemolysis. They can also act as opsonins and cause extravascular hemolysis.

(i) Autoantibody mechanism

Drugs like α-methyl dopa induce autoantibody in an unknown manner, particularly Rh blood group antigens.

HEMOLYTIC ANEMIA DUE TO INFECTIONS

Several organisms can cause hemolysis by different mechanisms. Some like Plasmodium and Babesia colonize RBCs and cause hemolysis and yet others like Bartonella, Clostridium adhere to cell surface and such cells are removed by

the spleen. Other mechanisms involved maybe the action of antibodies against antigen coated cells leading to agglutination [104] or to complement mediated lysis[105] .

UNEXPLAINED ANEMIA

The traditional notion that anemia in geriatric patients signifies some serious underlying disorder is not always true. A proportion of elderly have anemia that does not meet any criteria for any etiology. Even with the advent of better tests, such as serum ferritin , methylmalonic acid, and soluble transferrin receptor, a significant portion of elderly persons with anemia will be diagnosed as having unexplained anemia[106].The pathophysiology of unexplained anemia is poorly understood and is primarily a diagnosis of exclusion. Whether unexplained anemia represents a spectrum of undiagnosed diseases is unclear. Several theories have been postulated to explain this phenomenon, including decreased production of hematologic factors, inflammatory cytokine presence, marrow abnormalities and androgen deficiencies[107].

CONSEQUENCES OF ANEMIA IN GERIATRIC PATIENTS:

Morbidity and mortality related to anemia in the geriatric age group can occur from underlying disease or due to effects of the anemia itself and has the following consequences:

MUSCLE AND BONE

Anemia and osteoporosis are both prevalent in the geriatric population with a negative relationship between low hemoglobin levels and bone mass. Also lower

hemoglobin levels are associated with lower ankle extension strength, lower muscle density and less muscle mass.

MOBILITY

Anemia has been associated with greater average decline in physical performance[108,109,110].The risk of mobility problems in persons with hemoglobin< 12 mg/dl is more than twice than that occurring in persons with hemoglobin > 13 mg/dl.

FALLS

Thirty percent of people over the age of 65 years fall at least once a year. Falls and fractures are significant causes of disability, admission to institutional care and mortality. Hemoglobin level less than 10g/dl is associated with a higher incidence of fall and injury to hip and head as compared to those who were not anemic[110, 111, 112].

COGNITION

Anemia due to vitamin B 12 deficiency and anemia alone has been demonstrated to be a risk factor for functional and cognitive decline[113].

CARDIAC DISEASE

Acute and chronic anemia can cause congestive cardiac failure in patients without heart disease and may precipitate congestive heart failure and angina in patients with underlying heart disease[114].

HOSPITAL ADMISSIONS

Anemia in elderly has been associated with frequent hospital admission for various causes[115].

HISTORY AND PHYSICAL EXAMINATION:

MEDICAL HISTORY:

Recent hospitalization can cause anemia in the elderly as a result of multiple phlebotomies as well as the acute illness itself [116, 117]. Any history of recent surgery leads one to think of acute blood loss, also when the person needs transfusion during surgery it implicates the person was anemic before the surgery and the presence of another condition preventing an appropriate response to blood loss should be thought of. Diseases that cause anemia including malignancies, myelodysplastic syndrome, chronic kidney disease and rheumatologic diseases should be taken into account. Dietary history is important since strict vegan diets can raise the risk of B-12 deficiency [118] and history of alcoholism should not be overlooked as it is a cause of B 12 and folate deficiencies[118].

History of previous transfusions is an indication of the presence of a chronic illness like thalassemia or other hemolytic diseases.

History of drug intake should be reviewed to rule out the drugs as a cause for anemia.

SYMPTOMS:

Most symptoms of anemia are non specific; however a temporal relation between falling hemoglobin and exacerbation of symptoms is very useful.

General symptoms include the following:

- Fatigue
- Weakness
- Exertional dyspnoea

- Tinnitus
- Presyncope
- Palpitations
- Poor concentration

Symptoms of iron deficiency:

- Blood loss- tarry stools, hematochezia, hematuria
- Pica
- Dysphagia
- Mouth and tongue soreness

Symptoms of vitamin B 12 deficiency

- Neuropathy
- Ataxia
- Dementia

Symptoms of hemolysis

- Jaundice
- Dark coloured of urine

PHYSICAL EXAMINATION:

Careful physical examination helps in revealing cause of the anemia hence justifying comprehensive physical examination. One should look for the following:

- Pallor (present in anemia due to any cause)(**image 1**)
- Icterus (seen in haemolytic anemia) (**image 2**)
- Koilonychia (in iron deficiency) (**image 3**)

- Lymphadenopathy (in metastasis due to malignancies) (**image 4**)
- Glossitis (**image 5**)
- Aphthous ulcers (**image 6**)
- Angular stomatis (**image 7**)
- Pedal edema (**image 8**)
- Tachycardia (in severe anaemia due to any cause)
- Cardiac murmurs (in severe anemia)
- Hepatomegaly (in haemolytic anemias)
- Splenomegaly (in haemolytic anemia)

LABORATORY ASSESSMENT OF ANEMIA:

After obtaining history and initial physical examination laboratory evaluation should be done which includes:

- Complete blood count
- Stool for occult blood
- Iron profile
- Vitamin profile
- Erythropoietin level
- Peripheral smear examination

COMPLETE BLOOD COUNT:

The CBC is important for the diagnosis of anemia and for monitoring disease progression and treatment efficacy. When assessing the elderly anemia patient, the most important components of the CBC are [119]:

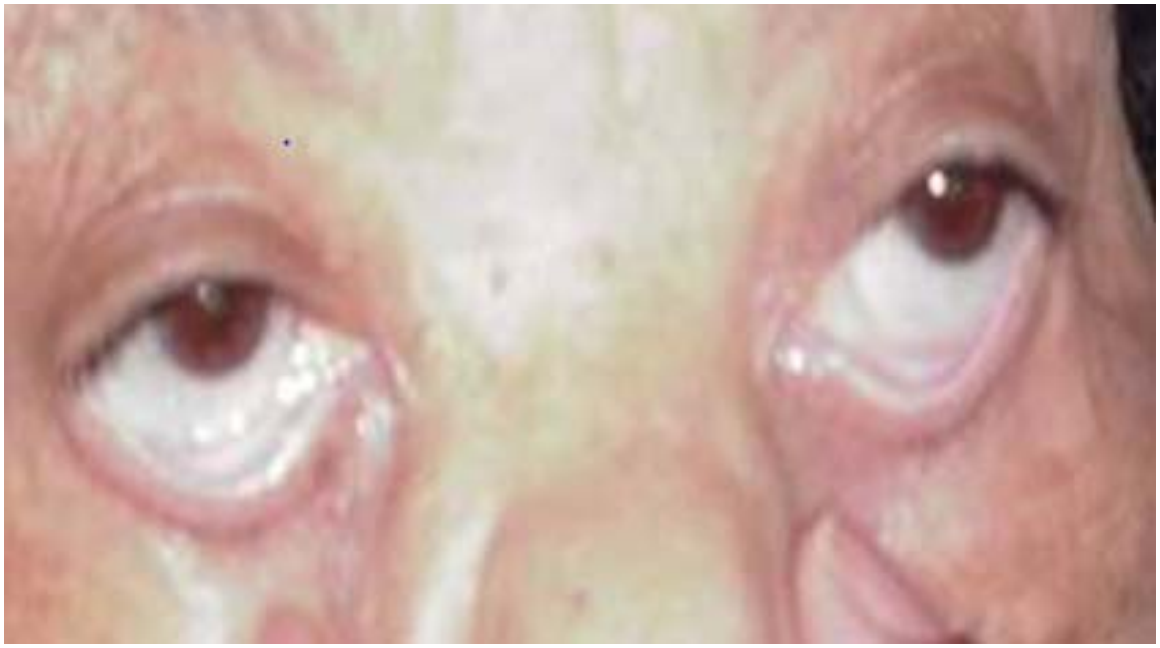


IMAGE 1. BULBAR CONJUNCTIVAL PALLOR

IMAGE 2. ICTERUS

IMAGE 3. KOILONYCHIA

IMAGE 4. LYMPHADENOPATHY

IMAGE 5. GLOSSITIS

IMAGE 6. APTHOUS ULCERS

IMAGE 7. ANGULAR STOMATITIS

IMAGE 8 PEDAL EDEMA

Erythrocyte (RBC) count:

Reports the total number of RBCs per litre of whole blood.

- Normal range for men: 4.7–6.1 million cells/mcL
- Normal range for women: 4.2–5.4 million cells/mcL

Hemoglobin (HgB):

Measures the amount of hemoglobin present in the blood

- Normal range for men: 13–17 g/dL
- Normal range for women: 12–16 g/dL

Hematocrit (HCT):

Packed cell volume in proportion to blood volume.

- Normal range for men: 40% to 52%
- Normal range for women: 36% to 48%

Mean cell (corpuscular) volume (MCV):

It measures the average size of RBCs, a diagnostic parameter for evaluating anemia, and differentiates microcytic and normocytic anemia in the elderly.

- Normal range: 81–100 fL
- Macrocytosis: Greater than 100 fL with large RBCs
- Microcytosis: Less than 81 fL with small RBCs

Mean cell hemoglobin (MCH):

It is the average amount of Hb in a RBC.

- Normal range: 27–34 Hb/cell

Mean cell hemoglobin concentration (MCHC):

It is the average concentration of Hgb in an RBC.

- Normal range: 30% to 36%

RBC distribution width (RDW-CV):

It measures variations in the size of RBCs.

- Normal range: 12% to 14%

Leukocyte (white blood cell) count:

Reports the number of leukocytes in the blood; the differential includes different types of leukocytes (i.e., neutrophil, eosinophil, basophil, lymphocyte, monocyte).

- Normal range: 4,500–10,000 cells/ mcL

Thrombocytes /platelet count:

It indicates the number of platelets present.

- Normal range: 150,000–450,000 cells/ mcL

STOOL EXAMINATION:

Stool specimen for occult blood is another essential test in evaluating anemia in elderly as GI bleed is often a major etiologic factor. The bleed may be intermittent so three stool samples testing is recommended. Before the test, the patient is advised to stop iron supplements if any, to avoid red meat and food with red dye for 3 days before the test.

ENDOSCOPIC EVALUATION:

If the patient tests positive for occult blood in the stool sample a gastroenterology consult is ideal. The patient will need endoscopy or colonoscopy to identify the bleed. It might indicate haemorrhoids or less other serious problems, it might also indicate malignancy.

IRON PROFILE:

Serum iron is not an adequate test to exclude iron deficiency. Numerous tests are available to diagnose iron deficiency including RBC, MCV, serum iron, serum transferrin, iron saturation, serum ferritin, soluble transferrin receptor (sTFR).

Serum ferritin is the most useful test for diagnosing iron deficiency. Low serum ferritin is highly specific for iron deficiency [120].As an acute phase reactant, ferritin can be elevated in inflammation, complicating the diagnosis of iron deficiency anemia in the presence of inflammation[121].Values between 18 and 44 ng/ml are highly suggestive of iron deficiency in the elderly[122, 123].

Alternative strategies to diagnose iron deficiency anemia have been studied, most commonly employing the sTFR. The sTFR is a truncated fragment of the membrane receptor that is increased in iron deficiency, when iron availability for erythropoiesis is low. STFR enhances diagnostic sensitivity in elderly persons compared with using a very low serum ferritin for iron deficiency anemia[124].-Another method standardizes sTFR based on the serum ferritin. By using the log of the sTFR/ferritin, a ratio greater than 2.5 may indicate iron deficiency. However, raising the threshold of ferritin to less than 30-50 ng/mL

affords a similar or better diagnostic performance.[123, 125] Further, sTFR is less sensitive in detecting early iron deficiency compared with ferritin.[126]

The iron profile will measure:

Total serum iron

- Normal range for men: 60–176 mcg/dL
- Normal range for women: 45–170 mcg/dL

Total iron binding capacity

- Normal range: 250–450 mcg/dL

Unsaturated iron binding capacity

- Normal range: 100–400 mcg/dL

Transferrin saturation

- Normal range: 20% to 50%

Serum ferritin

- Normal range for men: 12–350 ng/mL
- Normal range for women: 12–200 ng/mL

VITAMIN PROFILE:

The vitamin profile may include measurements of:

Folate (vitamin B9)

- Normal range: 4–20 mcM

Vitamin B12 (cobalamin)

- Normal range: 200–900 pg/mL

Methylmalonic acid

- Normal range: 73–271 nM

Homocysteine

- Normal range: 5.1–13.9 mcM

Vitamin B 12 and folate deficiencies though rare are essential to rule out as a cause of anemia as these are reversible causes. Low vitamin B-12 levels are frequent in older adults. If a level less than 200 pg/mL is detected, then cause of vitamin B-12 deficiency (eg, pernicious anemia, malabsorption) should be investigated and vitamin B-12 replenished. For equivocal levels of vitamin B-12, such as a level between 200 and 350 pg/mL, evaluate methylmalonic acid. An elevated methylmalonic acid level is evidence of a vitamin B-12 tissue deficiency. One must recognize that methylmalonic acid is also elevated in renal dysfunction. Homocysteine is elevated in vitamin B-12 deficiency and folate deficiency. If the methylmalonic acid is elevated in the setting of anemia and a low vitamin B-12, evaluate for vitamin B-12 deficiency and empirically treat the patient.

ERYTHROPOIETIN LEVELS:

Erythropoietin level is not ordered frequently, but it is used in patients with unexplained polycythemia which is a condition with abnormally elevated concentration of RBCs. The erythropoietin level is also reduced in chronic kidney disease and myelodysplasia. The normal range is 4.5–21.3 mU/mL[127]. Measuring serum erythropoietin levels is of little diagnostic utility in patients with chronic kidney disease.

PERIPHERAL SMEAR EXAMINATION:

Peripheral blood smear is essential to determine the caue of anemia. The peripheral enables one to study the morphology of the three cell components and is highly subjective but highly rewarding.

MORPHOLOGY OF RED BLOOD CELLS

In a well spread dried and stained blood film the RBC have round smooth contours and diameters between 6.0 – 8.5 μm[128] with a central pallor approximately one third of the area of the red blood cell.

Anisocytosis and poikilocytosis

These terms mean more variation in size (anisocytosis) or shape (poikilocytosis) (**image 9**) than that usually expected in a smear. It is seen in iron deficiency anemia, megaloblastic anemia, thalassemias and myelodysplastic syndromes.

Macrocytes

Macrocytes are present classically in megaloblastic anemias[128] and also in aplastic anemia, myelodysplastic syndromes and dyserythropoietic conditions.

Microcytes

Microcytes in a peripheral smear (**image 10**) occur in defects of hemoglobin synthesis[128] and are seen in iron deficiency, thalassemia, severe cases of anemia of chronic disease.

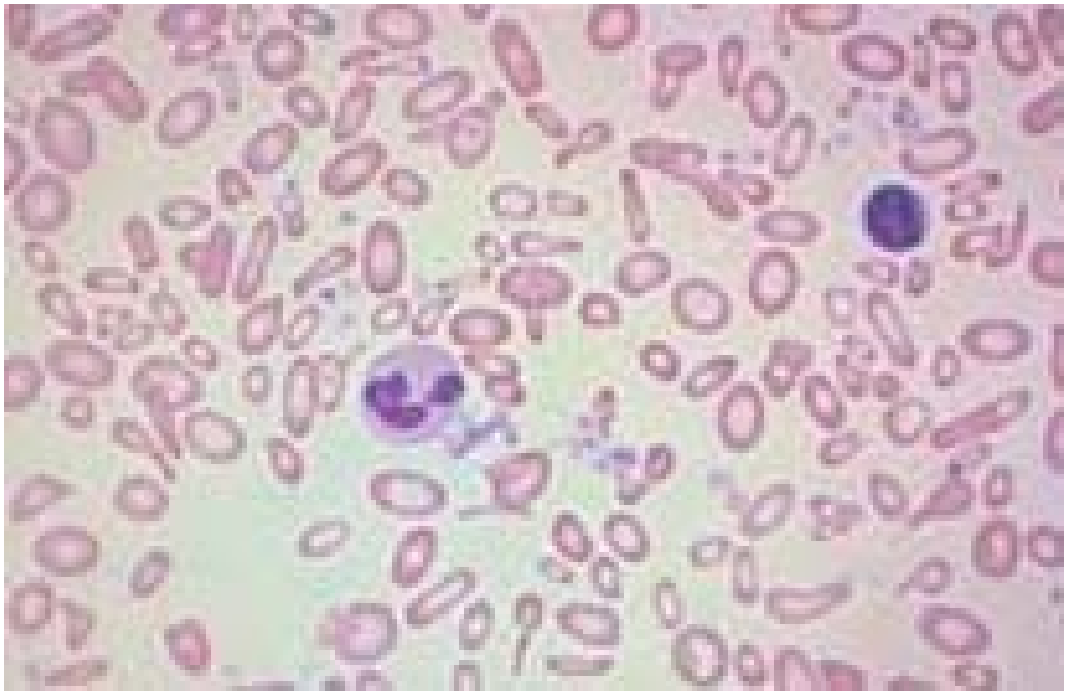


IMAGE 9. Photomicrograph showing peripheral smear with Anisopoikilocytosis (1000x, oil, leishman stain)

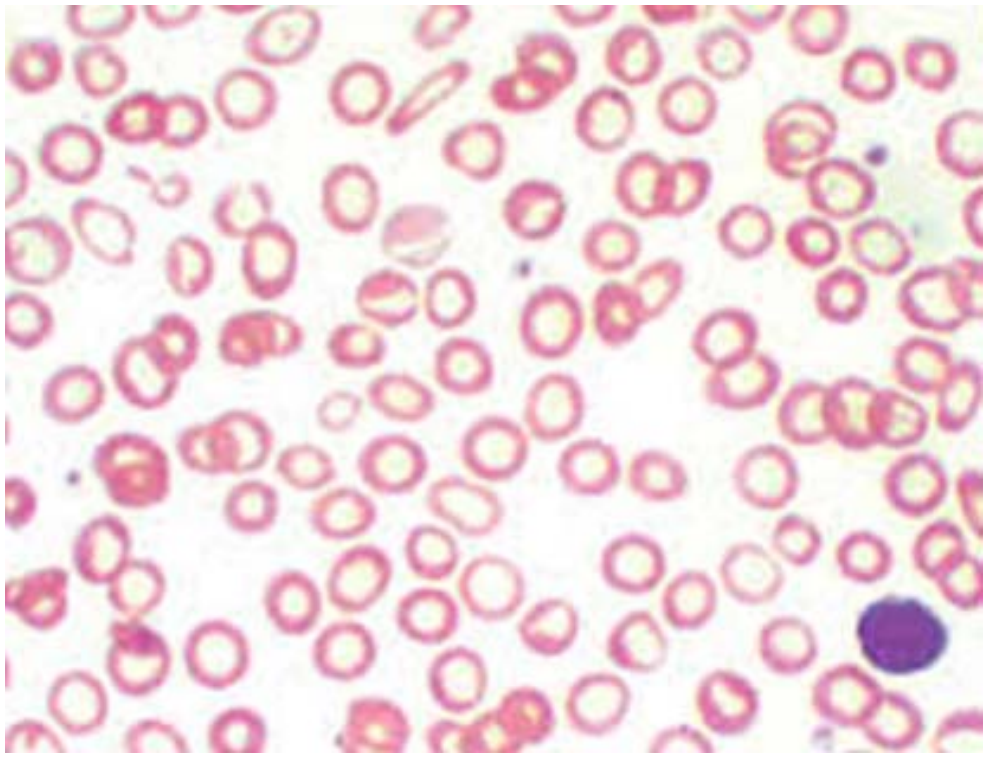


IMAGE 10. Photomicrograph showing peripheral smear with Microcytic Red Blood Corpuscles (1000x, oil, leishman stain)

Basophilic stippling

The presence of basophilic granules that are distributed throughout the cell is termed basophilic stippling and is seen in conditions like thalassemia, liver disease, lead poisoning and megaloblastic anemias.

Hypochromia

Occurrence of the central pallor more than one third of the area of the cell helps one to identify hypochromia which occurs in conditions of reduced hemoglobin synthesis or unusually red cells. Iron deficiency anemia and sideroblasticanemias are some of causes of hypochromia.

Hyperchromasia

Unusually deep staining of cells with loss of central pallor is seen when theres presence of macrocytes and when are rounded[128]. It is seen in megaloblastic anemias and spherocytosis.

Schistocytosis

Schistocytes are red cell fragments that are smaller than normal red cells with irregular or round contour (**image 11**) and with hypochromasia or hyperchromasia. It can occur in conditions like thalassemias, congenital dyserythropoietic anemia, microangiopathic hemolytic anemia, cardiac hemolytic anemia and in thermal injury like burns.

Acanthocytosis

This describes red cells with irregular spicules of varying length, thickness and irregularly distributed over the cell surface and seen in abnormal phospholipid metabolism[129] and in liver diseases[130]

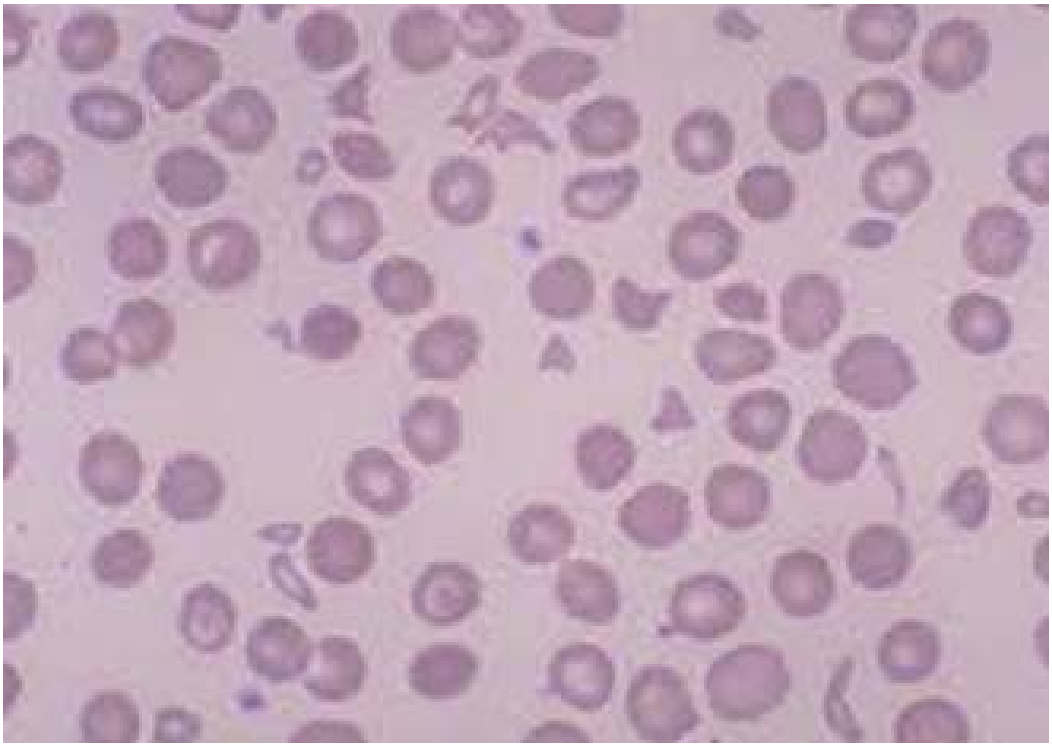


IMAGE 11. Photomicrograph showing peripheral smear with Schistocytes (1000x, oil, leishman stain)

Target cells

This term refers to cells with centrally stained area and a peripheral rim of hemoglobinized cytoplasm separated by non stained or lightly staining cytoplasm and is present in thalassemias, hereditary hypolipoproteinemia and some cases of iron deficiency anemia.

Sickle cells

Sickle cells are present in freshly drawn samples of blood from individuals homozygous for hemoglobin S and can be observed when subjected to hypoxia.

Howell-jolly bodies

Howell-jolly bodies are round cytoplasmic inclusions that stain purple with Romanowsky stains and are regularly present following splenectomy[128]

MORPHOLOGY OF LEUCOCYTES

Neutrophils

Normal neutrophils are 13μm in size with segmented nucleus having 2- 5 lobes connected by tapering chromatin strands and cytoplasm shows fine azurophilic granules when stained with Romanowsky stains. The nuclear chromatin is peripherally condensed with clumping.

Lymphocytes

The circulating lymphocytes are uniform in size about 9 μm in diameter with a thin rim of cytoplasm occasionally showing scant azurophilic granules.

Eosinophils

They are of size 12- 17 μm with a bilobed nucleus usually and have bright orange granules in the cytoplasm.

Monocytes

Monocytes are the largest of circulating leucocytes of size 15- 18 μm with blue- grey cytoplasm having variable number of reddish granules and large curved nucleus that maybe folded or curved.

Basophils

They are the least number of leucocytes with variably sized dark blue granules in the cytoplasm that tend to obscure the segmented nucleus.

BONE MARROW STUDY

The absolute indications for bone marrow examination include evaluation of leucopenia, anemia, pancytopenia, leucoerythroblastosis and diagnosis of leukemia as well as monitoring the effects of therapy[131,132]. Relative indications include evaluation of iron metabolism unexplained fever (PUO) or splenomegaly, and sampling for chromosomal analysis, immunophenotyping, or microbiological cultures[131, 132].

Iron deficiency anemia

The bone marrow in iron deficiency is characterized by mild to moderate erythroid hyperplasia with striking nuclear distortions, resembling those found in dyserythropoietic anemias[133]. Karyorrhexis and nuclear budding are particularly common, but multinuclearity, nuclear fragmentation, and even intranuclear bridging may be observed. The individual normoblasts appear small and may have scanty cytoplasm, often with irregular, ragged borders. Macrophage iron is absent or severely reduced in the marrow, spleen, and liver of iron-deficient subjects.

Megaloblastic anemia

Aspirated marrow is cellular with striking megaloblastic changes, especially in the erythroid series. The high apoptosis rate of erythroid precursor cells in the marrow creates more globin lysis and, thus, jaundice. Sideroblasts are increased in number and contain increased numbers of iron granules. The ratio of myeloid to erythroid precursors falls to 1:1 or lower, and granulocyte reserves may be decreased.

Hemolytic anemia

The marrow shows hyperplasia of the erythroid series with normoblasts constituting 25 -60 % of all nucleated cells.

Anemia of chronic inflammation

The marrow in cases of anemia with chronic inflammation is moderately cellular with erythroid hyperplasia and normal erythroid maturation.

Myelodysplastic syndrome

Marrow in myelodysplastic syndromes may be either normocellular or hypercellular [134,135,136]. The erythroid series may show large or small erythroblasts, nuclear fragmentation and stippled erythroblasts[134, 135, 136]. Granulocytic precursors show hypogranulation, monocytoid appearing granulocytes and Pelger-Huet anomaly of the neutrophils. Megakaryocytes appear micro megakaryocytes with unilobed or bilobed nuclei[134, 135, 136]

Myelophthisic anemia

Marrow aspirate is usually unyielding and marrow biopsy reveals clusters of large anaplastic cancer cells bearing features of the primary tumour and lymphocytes in cases of lymphoproliferative disorders can be present

MATERIALS AND METHODS

STUDY LOCATION

Departments of General medicine, General surgery and Pathology, Tirunelveli Medical College Hospital, Tirunelveli – 11.

PERIOD OF STUDY

2 years between September 2013 and September 2015

SIZE OF SAMPLE

100 geriatric patients (> 65 YEARS OLD) with clinical features of anemia.

TYPE OF STUDY

Hospital based observational study

INCLUSION CRITERIA

1. Patients above the age of 65 years
2. Men with hemoglobin levels less than 13 mg/dL and women with hemoglobin less than 12 mg/dL

EXCLUSION CRITERIA

1. Patients below age of 65 years
2. Patients who refused to consent to take part in the study

3. Patients who refused to consent for venipuncture for collection of blood samples.

METHODOLOGY

The cases received in the hematology department for peripheral smear reporting were screened and 100 cases that showed hemoglobin levels lower than standard WHO values were chosen. The entire medical history was obtained from each of the cases including previous medical reports and imaging studies. Blood samples were collected from all patients in plain vacutainers and EDTA containing vacutainers. Complete blood counts for all the samples was done using SYSMEX 3 part differential analyzer and the RBC indices was noted along with total WBC count, differential count, platelet count and hematocrit.ESR for all cases was done using Westergren tube. Peripheral smears were made for all the samples and stained using Leishman stain. Supravital staining using methylene blue was done for reticulocyte counting. Ferritin, iron and TIBC for all samples was done using SPINREACT kits for all the three by the following methods.

FERRITIN

The patients' blood was collected in plain vacutainers and serum separated by centrifuging at 3000 rpm / min for 5 minutes. The tubes that were used to run the ferritin assay were prewarmed to 37• C before running the test. 400 µL of the R1 buffer (provided with the kit) was added to the prewarmed tube followed by 100 µL of R2 latex buffer (provided with the kit), then 50 µL of the patient's serum was added. The result is then read using turbidometry at an absorbance of

540 nm. The values were standardized using control sera and fixed between 30 and 220 µg/ L for men and between 20 and 110 µg/L for women

IRON

The assay for iron was run using a blank for every sample. The pair of tubes were prewarmed to 37• C to which was added 500µL of R1 solution (working solution to which R2 was already mixed), 100 µL patient's serum, 100 µL distilled water were added. To the test tube 25 µL R3 was added whereas in the blank this step was avoided. The tubes were then incubated at room temperature for 10 minutes and the results were obtained using colorimetry at an absorbance of 570 nm. The test was standardized using control sera and normal range was set between 65 and 175 µg/L for men and between 40 and 150 µg/L for women

TOTAL IRON BINDING CAPACITY

To prewarmed test tube 250 µL of patient serum was added followed by 500 µL of R5 solution. The mixture was incubated at room temperature for 10 minutes after which 1 ½ spoonfuls of R6 reagent was added. This mixture was centrifuged at 3000 rpm for 15 minutes following 10 minutes incubation and the supernatant was collected and iron measured using previously said method. The standard value was set between 200 and 400 µg/dL for both men and women.

RESULTS AND OBSERVATIONS

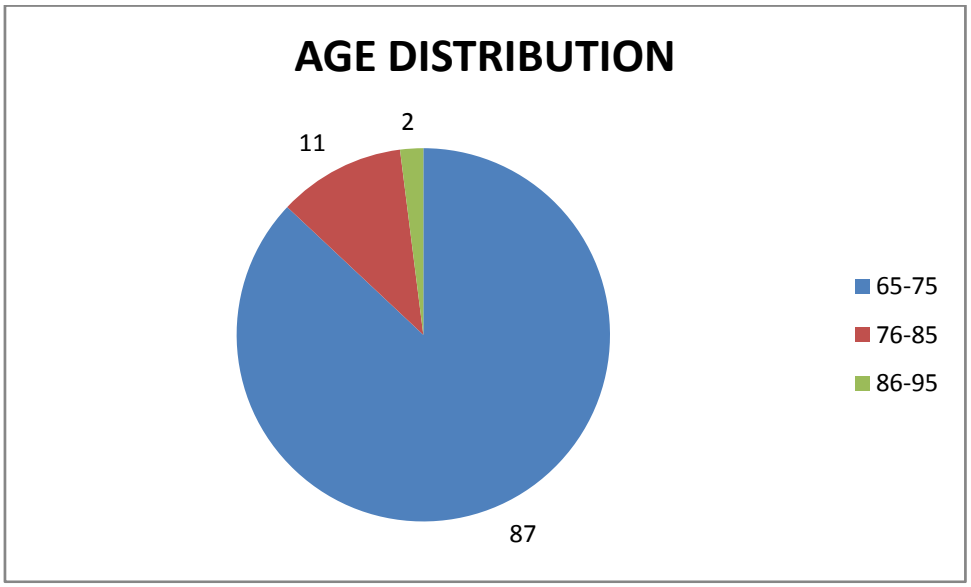
AGE DISTRIBUTION

Of the 100 patients studied 87 patients [87 %] fall in the age group of 65 to 75 years, 11 patients [11%]fall in the age group 0f 76 – 85 years and 2 patients fall in the age group of 86-95 years.

TABLE NO.2. AGE DISTRIBUTION

AGE	NO.OF PATIENTS	PERCENTAGE
65-75yrs	87	87
76-85yrs	11	11
86-95yrs	2	2

FIGURE NO.2. AGE DISTRIBUTION



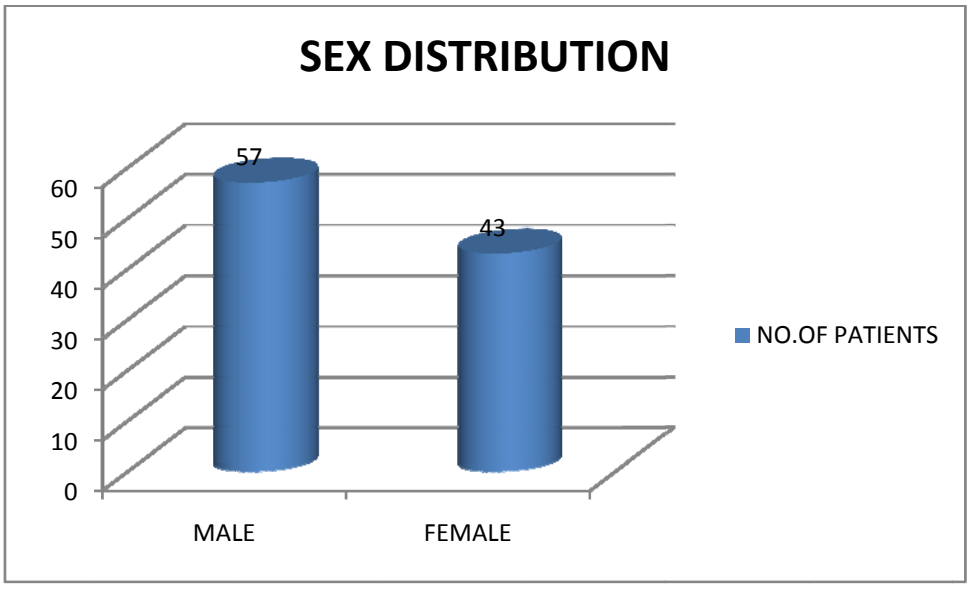
SEX DISTRIBUTION

Of the 100 cases studied 57 patients [57%] were men and 43 patients [43%] were women.

TABLE NO. 3. SEX DISTRIBUTION

SEX	NO.OF PATIENTS	PERCENTAGE
MALE	57	57
FEMALE	43	43

FIGURE NO. 3. SEX DISTRIBUTION



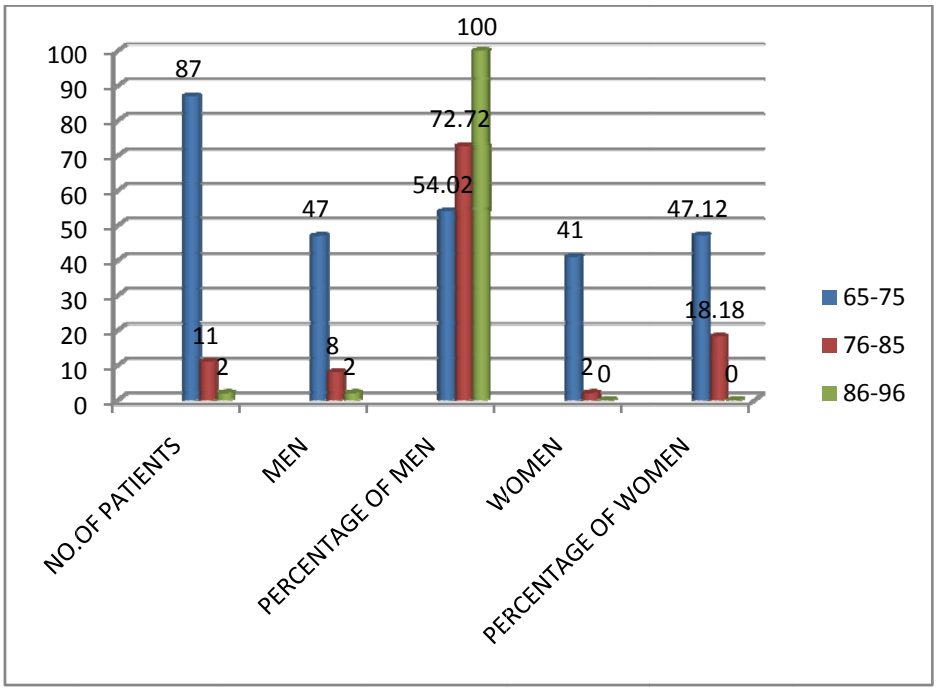
SEX DISTRIBUTION IN DIFFERENT AGE GROUPS

Of the 87 patients in the age group of 65 – 75, 47 were men (54.02%) and 41 were women (47.12 %). In the 11 patients in the age group of 76-85 years 72.72 % were men (8 patients) , 18.18 % were women and in the age group of 86 – 96 years 100 % were men.

TABLE NO. 4. SEX DISTRIBUTION IN DIFFERENT AGE GROUPS

AGE	NO.OF PATIENTS	MEN	PERCENTAGE OF MEN	WOMEN	PERCENTAGE OF WOMEN
65-75	87	47	54.02	41	47.12
76-85	11	8	72.72	2	18.18
86-96	2	2	100	0	0

FIGURE NO.4. SEX DISTRIBUTION IN DIFFERENT AGE GROUPS



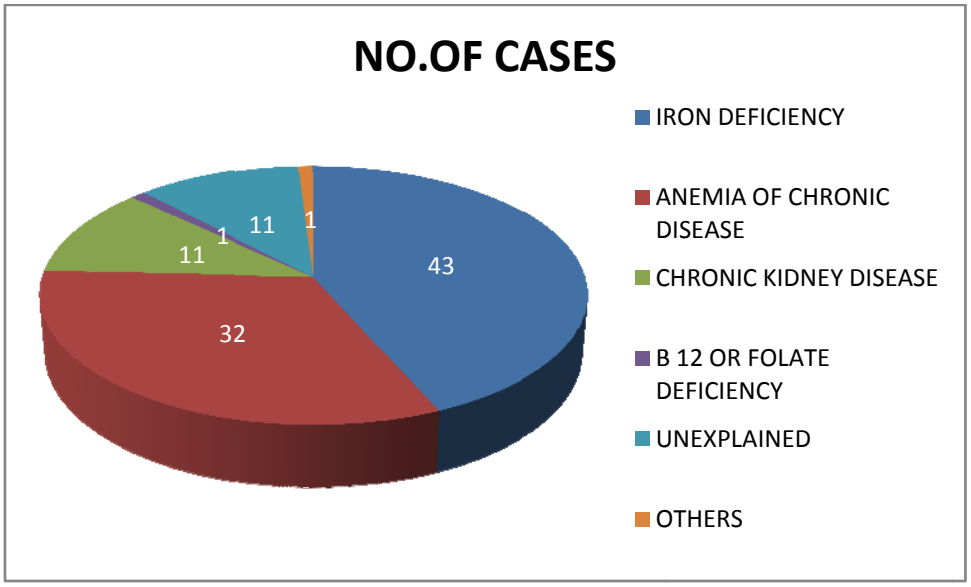
ETIOLOGY OF ANEMIA

Among the causes for anemia iron deficiency was the commonest constituting 43% of the cases followed by anemia of chronic diseases which constituted 32 % of the cases. Among the causes for anemia due to chronic inflammation 4 patients had both diabetes and hypertension, 2 had pulmonary tuberculosis, 2 had coronary heart disease, 1 was a case of thymoma, 2 were hematological malignancies, 2 cases were malignant melanomas, 4 were lung cancers, 3 breast cancers, 3 GI malignancies, 1 was a bone tumour, and 2 were unidentified malignancies. The next common cause for anemia was anemia of chronic kidney disease (11%) followed by anemia of unknown etiology (10%).

TABLE NO. 5. ETIOLOGY OF ANEMIA

DIAGNOSIS	NO.OF CASES	PERCENTAGE
IRON DEFICIENCY	43	43
ANEMIA OF CHRONIC DISEASE	32	32
CHRONIC KIDNEY DISEASE	11	11
B 12 OR FOLATE DEFICIENCY	1	1
UNEXPLAINED	11	11
OTHERS	1	1

FIGURE NO. 5. ETIOLOGY OF ANEMIA



INTENSITY OF ANEMIA

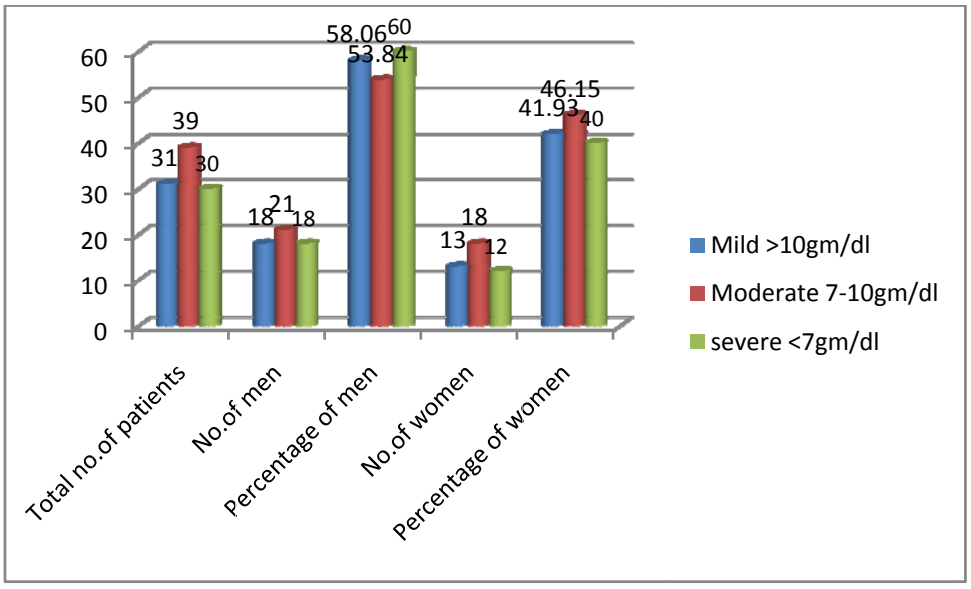
Based on the WHO criteria anemia can be classified into mild anemia with hemoglobin less than 10 gm/dl, moderate anemia with hemoglobin between 7 and 9.9 gm/dl and severe with hemoglobin less than 7 gm/dl [137].

Of the 100 cases studied 31 had mild anemia out of which 18 were men and 13 women, 21 had moderate anemia of which 21 were men and 18 were women and 18 had severe anemia of which 18 were men and 12 were women.

TABLE NO. 6. INTENSITY OF ANEMIA

	Hgb levels	Total no.of patients	No.of men	Percentage of men	No.of women	Percentage of women
Mild	>10gm/dl	31	18	58.06	13	41.93
Moderate	7-10gm/dl	39	21	53.84	18	46.15
severe	<7gm/dl	30	18	60	12	40

FIGURE NO.6. INTENSITY OF ANEMIA



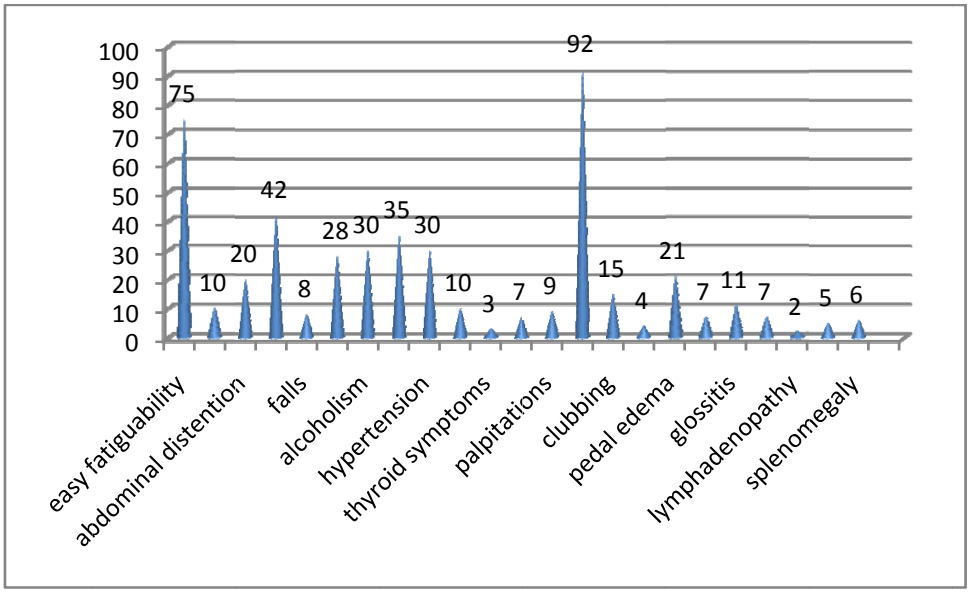
CLINICAL FEATURES

Of the 100 patients studied the commonest symptom associated with anemia was easy fatiguability which was present in 75 patients, followed by GI bleed in 10 patients and palpitation in 9 patients. Of the clinical signs pallor was commonest and was present 92% of patients, peripheral edema in 20 patients, tachycardia in 15 patients, glossitis in 11 patients, koilonychias in 7 patients, hepatomegaly in 5 patients and splenomegaly in 6 patients.

TABLE NO. 7. CLINICAL FEATURES OF ANEMIA

Easy fatiguability	75%
GI bleed	10%
Abdominal distention	20%
Chronic drug intake	42%
Falls	8%
Smoking	28%
Alcoholism	30%
Diabetes	35%
Hypertension	30%
Features of renal disease	10%
Thyroid symptoms	3%
Known malignancies	7%
palpitation	9%
Pallor	92%
Clubbing	15%
Icterus	4%
Pedal edema	21%
Koilonychia	7%
Glossitis	11%
Aphthous ulcer	7%
Lymphadenopathy	2%
Hepatomegaly	5%
Splenomegaly	6%
Tachycardia	15%

FIGURE NO.7. CLINICAL FEATURES OF ANEMIA



CORRELATION OF CLINICAL FEATURES WITH INTENSITY OF ANEMIA

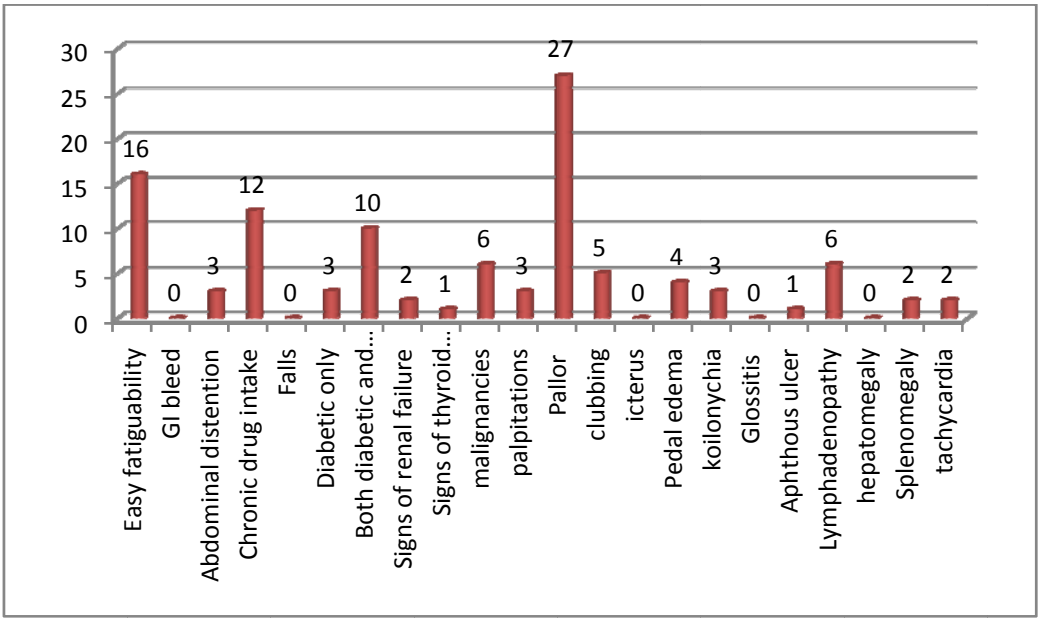
MILD ANEMIA

Of the 31 patients who presented with anemia only 16 patients complained of easy fatiguability, and there were no other specific complaints. 3 patients were diabetic, and 10 patients had both diabetes and hypertension and these 10 patients have history of chronic drug intake (oral hypoglycemic drugs and antihypertensives) , 2 patients had signs of renal failure and 6 were diagnosed with malignancy. 27 patients had pallor, 5 patients had clubbing, 3 patients had koilonychia, 2 patients presented with splenomegaly.

TABLE NO. 8. CLINICAL PROFILE OF PATIENTS WITH MILD ANEMIA

Easy fatiguability	16%
GI bleed	0%
Abdominal distention	3%
Chronic drug intake	12%
Falls	0%
Diabetic only	3%
Both diabetic and hypertensive	10%
Signs of renal failure	2%
Signs of thyroid disturbances	1%
malignancies	6%
palpitations	3%
Pallor	27%
clubbing	5%
icterus	0%
Pedal edema	4%
koilonychia	3%
Glossitis	0%
Aphthous ulcer	1%
Lymphadenopathy	6%
hepatomegaly	0%
Splenomegaly	2%
tachycardia	2%

FIGURE NO.8. CLINICAL PROFILE OF PATIENTS WITH MILD ANEMIA



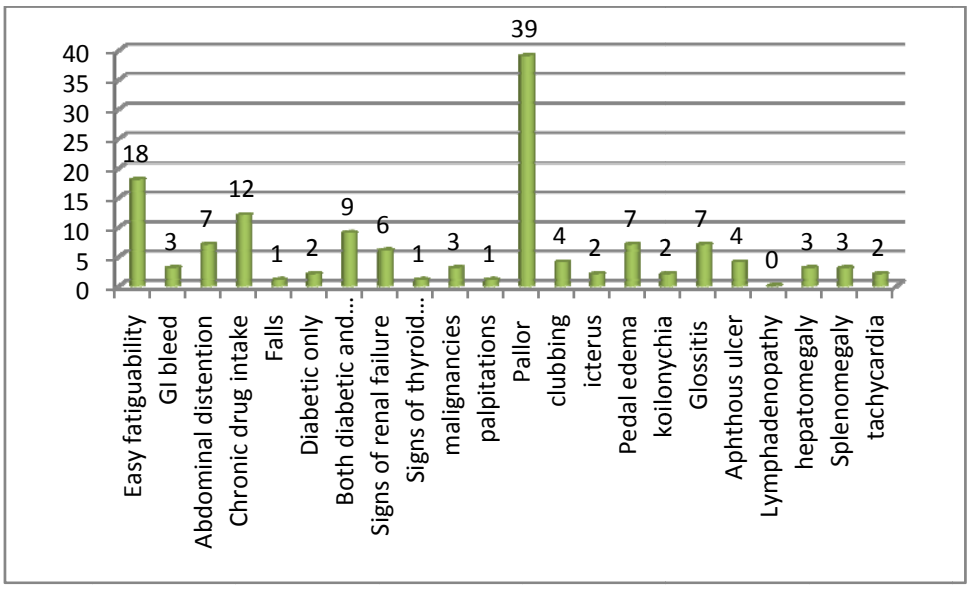
MODERATE ANEMIA

In our study 39 patients had moderate anemia out of which 18 patients had history of easy fatiguability, 3 had symptoms of GI bleed, 7 had abdominal distention, 12 had history of chronic drug intake, 2 patients were diabetic, 9 were both diabetic and hypertensive, 6 had symptoms of renal failure and 3 were diagnosed with malignancies. Of the 39 patients all had pallor, 2 patients had icterus, 7 had pedal edema, 2 had koilonychia, 7 had glossitis, 4 patients had aphthous ulcers, 3 had hepatomegaly, 3 had splenomegaly and 2 patients had tachycardia.

TABLE NO. 9. CLINICAL PROFILE OF PATIENTS WITH MODERATE ANEMIA

Easy fatiguability	18%
GI bleed	3%
Abdominal distention	7%
Chronic drug intake	12%
Falls	1%
Diabetic only	2%
Both diabetic and hypertensive	9%
Signs of renal failure	6%
Signs of thyroid disturbances	1%
malignancies	3%
palpitations	1%
Pallor	39%
clubbing	4%
icterus	2%
Pedal edema	7%
koilonychia	2%
Glossitis	7%
Aphthous ulcer	4%
Lymphadenopathy	0%
hepatomegaly	3%
Splenomegaly	3%
tachycardia	2%

FIGURE NO.9. CLINICAL PROFILE OF PATIENTS OF MODERATE ANEMIA



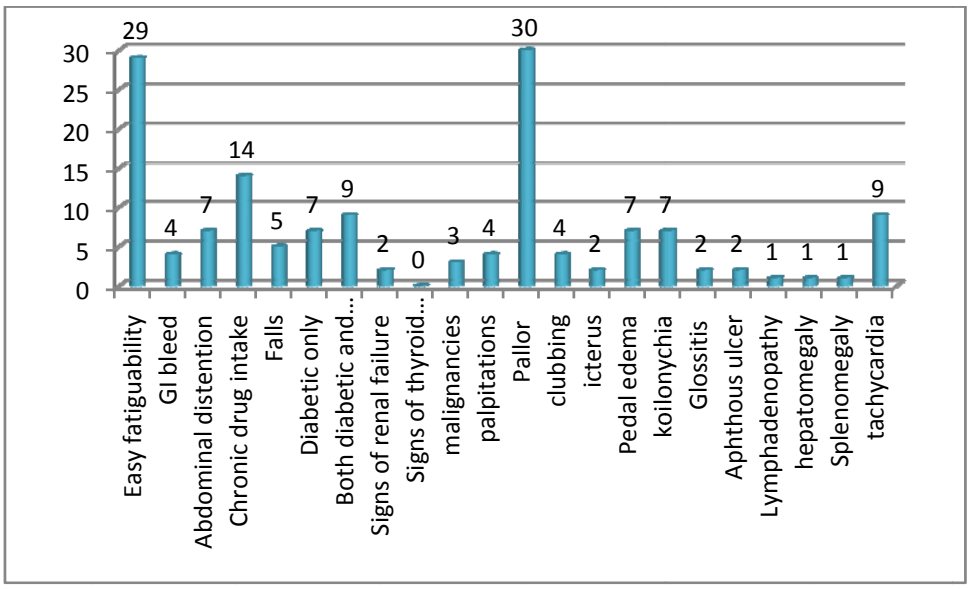
SEVERE ANEMIA

30 patients in our study had severe anemia. Of the 30, 29 patients had history of easy fatiguability, 4 had history of GI bleed, 7 had abdominal distention, 14 patients had history of chronic drug intake, 7 patients were diabetic alone, 9 patients had both hypertensive and diabetic, 2 had features of renal failure and 3 were diagnosed with malignancy. Out of the 30 patients all had pallor, 2 presented with icterus, 7 had pedal edema, 7 had koilonychias, 2 had glossitis, 2 had aphthous ulcers, 1 patient had lymphadenopathy, 1 had hepatomegaly, 1 had splenomegaly and 9 patients had tachycardia.

TABLE NO.10. CLINICAL PROFILE OF PATIENTS WITH SEVERE ANEMIA

Easy fatiguability	29%
GI bleed	4%
Abdominal distention	7%
Chronic drug intake	14%
Falls	5%
Diabetic only	7%
Both diabetic and hypertensive	9%
Signs of renal failure	2%
Signs of thyroid disturbances	0%
malignancies	3%
Palpitations	4%
Pallor	30%
Clubbing	4%
Icterus	2%
Pedal edema	7%
Koilonychias	7%
Glossitis	2%
Aphthous ulcer	2%
Lymphadenopathy	1%
hepatomegaly	1%
Splenomegaly	1%
Tachycardia	9%

FIGURE NO. 10. CLINICAL PROFILE OF PATIENTS WITH SEVERE ANEMIA



CORRELATION OF RED CELL INDICES WITH ETIOLOGY

The average levels of Hgb among the IDA, ACD, A- CKD and AUE was 7.7 gm/dl, 9.02gm/dl, 9.02 gm/dl and 8.9 gm/dl respectively. The average of MCV, MCH and MCHC in IDA was found to be 74.13fl, 24.47 hb/cell and 30.93 % wheras in Anemia of chronic disease it was seen to be 81.13 fl, 27.87 pg and 34.59 %. In A-CKD average MCV was 84.92 fl, MCH was 28.35pg and MCHC was 33.61 % and in AUE it was 80.01 fl, 25.16 pg and 31.49%

TABLE NO.11 CORRELATION OF RED CELL INDICES WITH ETIOLOGY

	Hgb (gm/dl)	MCV (fl)	MCH (pg/cell)	MCHC (%)
IDA	7.7	74.13	24.47	30.93
ACD	9.02	81.13	27.87	34.59
CKD	7.93	84.92	28.35	33.6
AUEO	8.9	80.01	25.16	31.49

FIGURE NO.11 CORRELATION OF RED CELL INDICES WITH ETIOLOGY

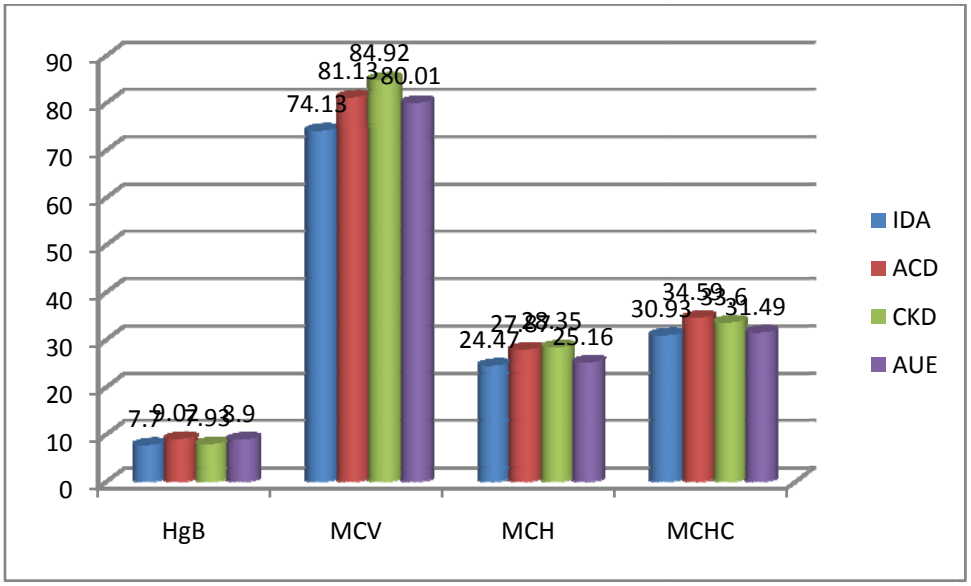
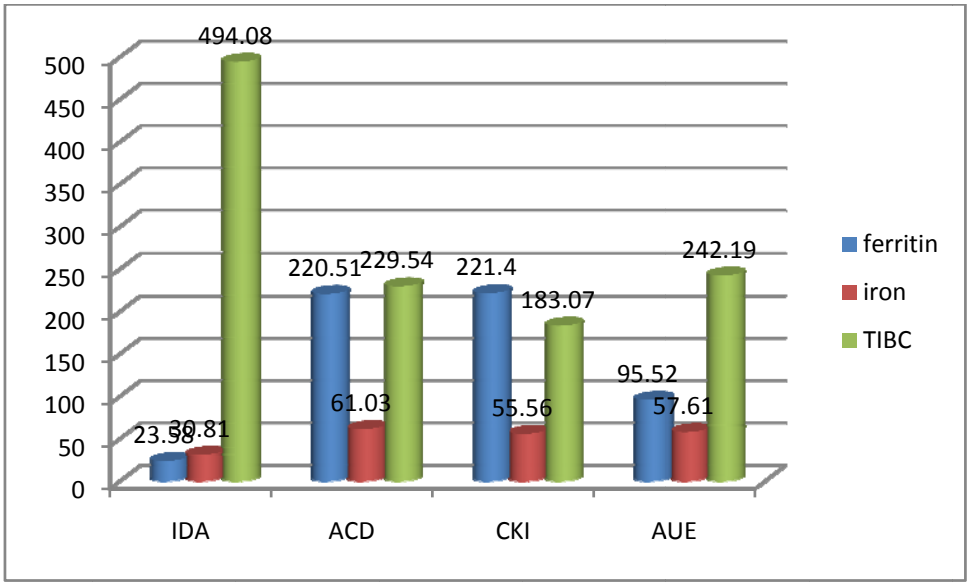


TABLE NO.12 CORRELATION OF RED CELL INDICES WITH ETIOLOGY OF ANEMIA

	Ferritin (µg/L)	Iron (µg/L)	TIBC
IDA	23.58	30.81	494.08
ACD	220.51	61.03	229.54
CKI	221.4	55.56	183.07
AUE	95.52	57.61	242.19

FIGURE NO.12 .CORRELATION OF RED CELL INDICES WITH ETIOLOGY OF ANEMIA



CORRELATION OF RED CELL INDICES WITH INTENSITY OF ANEMIA

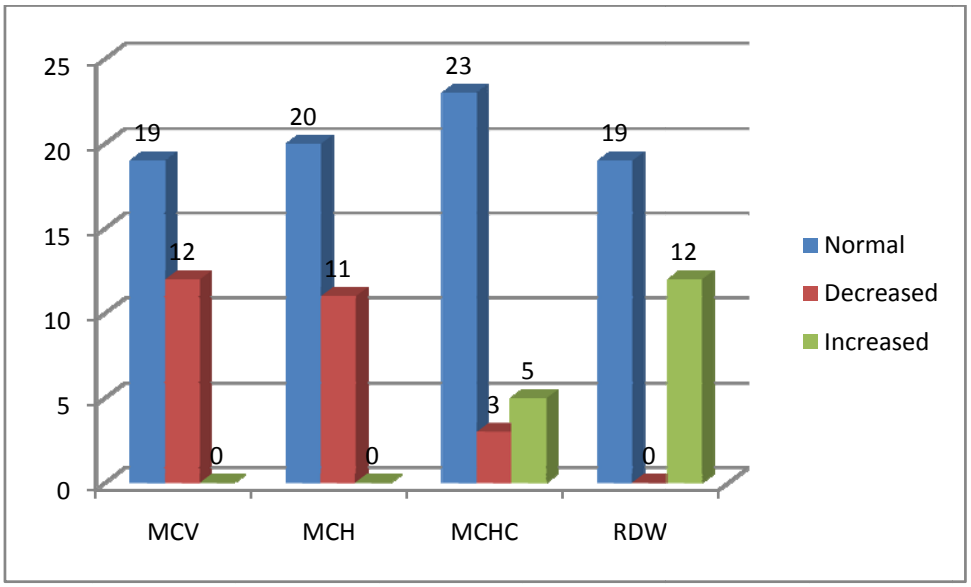
Of the 31 patients with mild anemia 19 patients had normal MCV, 20 had normal MCH, 23 patients had normal MCHC and 19 had normal RDW. Low MCH was found in 12 patients, low MCH in 11, low MCHC in 3 patients whereas increased

MCV or MCH was seen in none, increased MCHC was seen in 5 patients and increased RDW was found in 12 patients.

TABLE NO.13. RBC INDICES IN PATIENTS WITH MILD ANEMIA

	MCV	MCH	MCHC	RDW
Normal	19	20	23	19
Decreased	12	11	3	0
Increased	0	0	5	12

FIGURE NO.13 . RBC INDICES IN PATIENTS WITH MILD ANEMIA

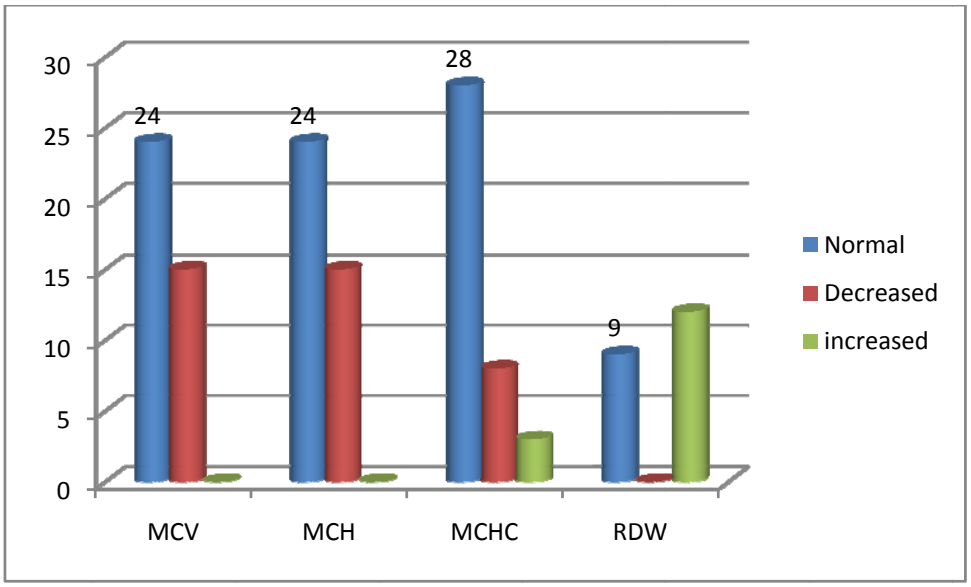


Out of 39 patients with moderate anemia normal MCV was present in 24 patients, normal MCH in 24 patients, normal MCHC in 23 patients and normal RDW was seen in 9 patients. 15 patients had decreased MCV, 15 had decreased MCH 8 had decreased MCHC, whereas 3 had increased MCHC and 30 had increased RDW.

TABLE NO.14. RBC INDICES IN MODERATELY ANEMIC PATIENTS

	MCV	MCH	MCHC	RDW
Normal	24	24	28	9
Decreased	15	15	8	0
increased	0	0	3	12

FIGURE NO.14. RBC INDICES IN MODERATELY ANEMIC PATIENTS

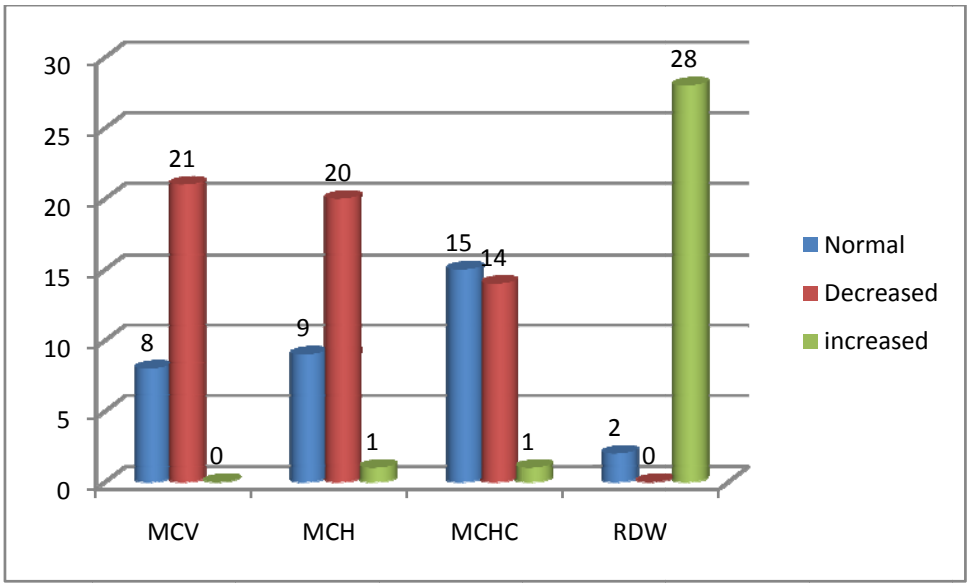


Among the 30 severely anemic patients normal MCV, MCH, MCHC and RDW was found in 8, 9, 15 and 2 patients respectively, low MCV, MCH, MCHC was found in 21, 20 and 14 patients respectively with high MCH, MCHC and RDW found in 1, 1 and 28 patients respectively.

TABLE NO.15. RBC INDICES IN SEVERELY ANEMIC PATIENTS

	MCV	MCH	MCHC	RDW
Normal	8	9	15	2
Decreased	21	20	14	0
increased	0	1	1	28

FIGURE NO.15. RBC INDICES IN SEVERELY ANEMIC PATIENTS



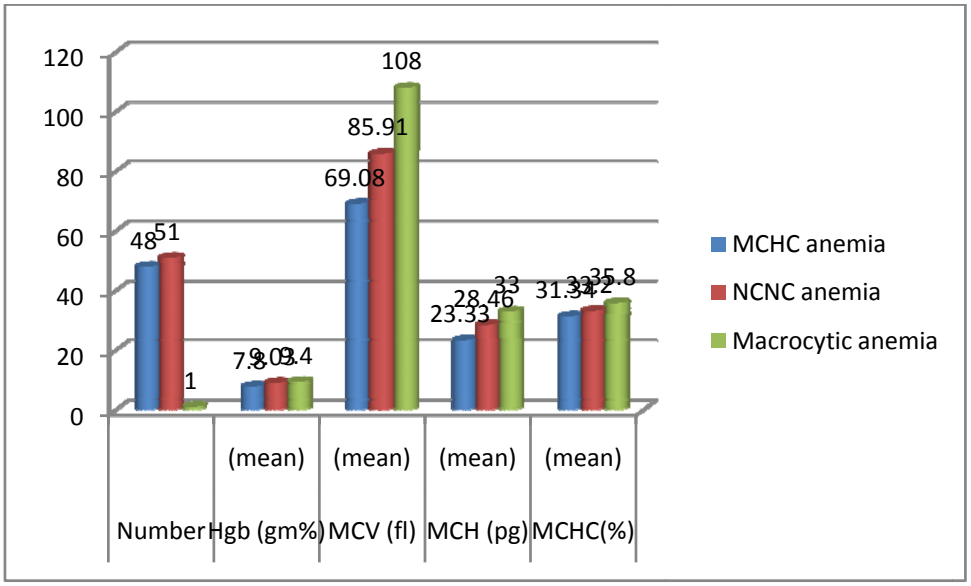
RBC INDICES IN VARIOUS TYPES OF ANEMIA

The mean values of RBC indices in microcytic hypochromic anemia is as follows: MCV- 69.08 fl, MCH 23.33 pg/cell, MCHC 31.34%, in normocytic normochromic anemia is as follows : MCV 85.91 fl, MCH 28.46 pg/cell, MCHC 33.2% and in dimorphic anemia is MCV 31.34 fl, MCH 33.2 pg/cell, MCHC 35.8%.

TABLE NO. 16. RBC INDICES IN VARIOUS TYPES OF ANEMIA

	Number	Hgb (gm%) (mean)	MCV (fl) (mean)	MCH (pg) (mean)	MCHC(%) (mean)
MCHC anemia	48	7.8	69.08	23.33	31.34
NCNC anemia	51	9.03	85.91	28.46	33.2
Macrocytic anemia	1	9.4	108	33	35.8

FIGURE NO.16. RBC INDICES IN VARIOUS TYPES OF ANEMIA



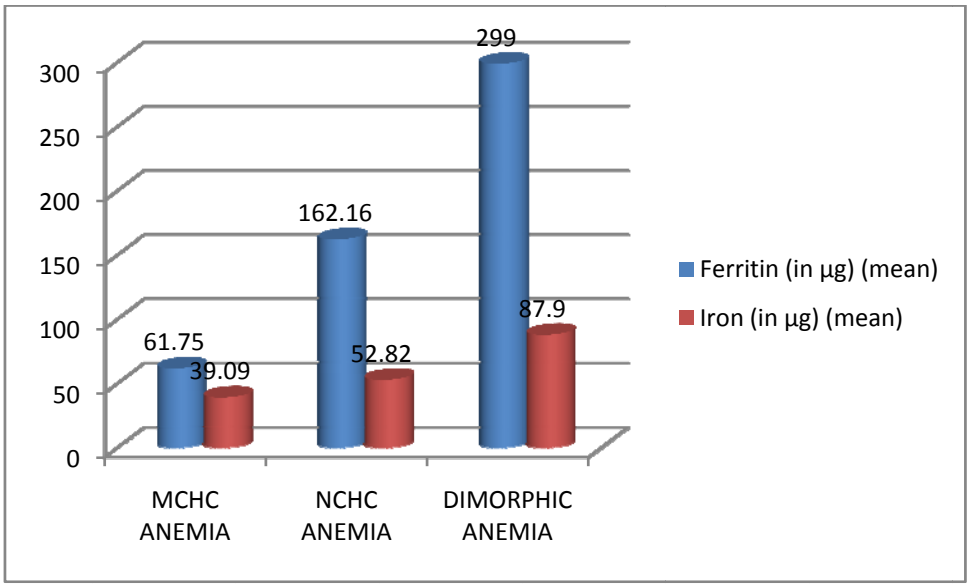
COMPARISON BETWEEN IRON PARAMETERS IN VARIOUS TYPES OF ANEMIA

The mean ferritin levels in microcytic hypochromic anemia in this study was found to be 61.75 and iron values was 39.09. The same indices in normocytic normochromic anemia was 162.16 and 52.82 and in dimorphic anemia was 299 and 87.9.

TABLE NO 17. IRON PARAMETERS IN VARIOUS TYPES OF ANEMIA

	MCHC anemia	NCHC anemia	dimorphic anemia
Ferritin (in µg) (mean)	61.75	162.16	299
Iron (in µg) (mean)	39.09	52.82	87.9

FIGURE NO. 17. IRON PARAMETERS IN VARIOUS TYPES OF ANEMIA



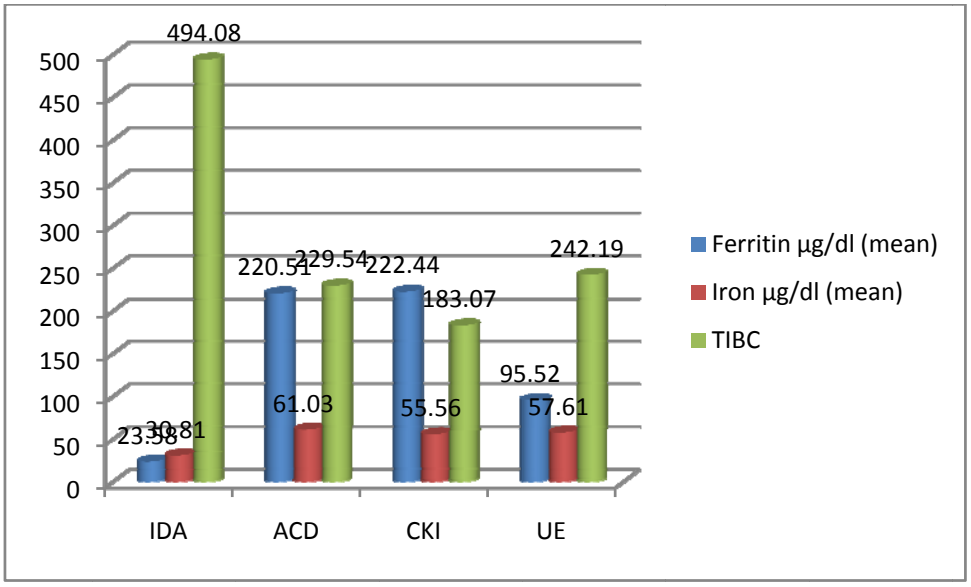
COMPARISON OF IRON PARAMETERS BASED ON ETIOLOGY OF ANEMIA

The ferritin values according to the various etiologies showed the following values in iron deficiency anemia it was 23.58 µg/dl, in anemia of chronic disease it was 220.51 µg/dl, anemia in chronic disease was 222.44µg/dl, unexplained anemia was 95.52µg/dl.

TABLE NO.18. IRON PARAMETERS BASED ON ETIOLOGY OF ANEMIA

	IDA	ACD	A-CKD	AUE
Ferritin µg/dl (mean)	23.58	220.51	222.44	95.52
Iron µg/dl (mean)	30.81	61.03	55.56	57.61
TIBC	494.08	229.54	183.07	242.19

FIGURE NO. 18. IRON PARAMETERS BASED ON ETIOLOGY OF ANEMIA



COMPARISON BETWEEN IRON PARAMETERS AND INTENSITY OF ANEMIA

Normal ferritin levels were seen in 20 mildly anemic patients, 19 moderately anemic patients and 30 severely anemic patients. Ferritin levels were low in 4 mildly anemic, 10 moderately anemic and 8 severely anemic patients whereas increased ferritn levels was seen in 7 in the mildly anemic group, 10 in moderately anemic group and 8 in severely anemic group.

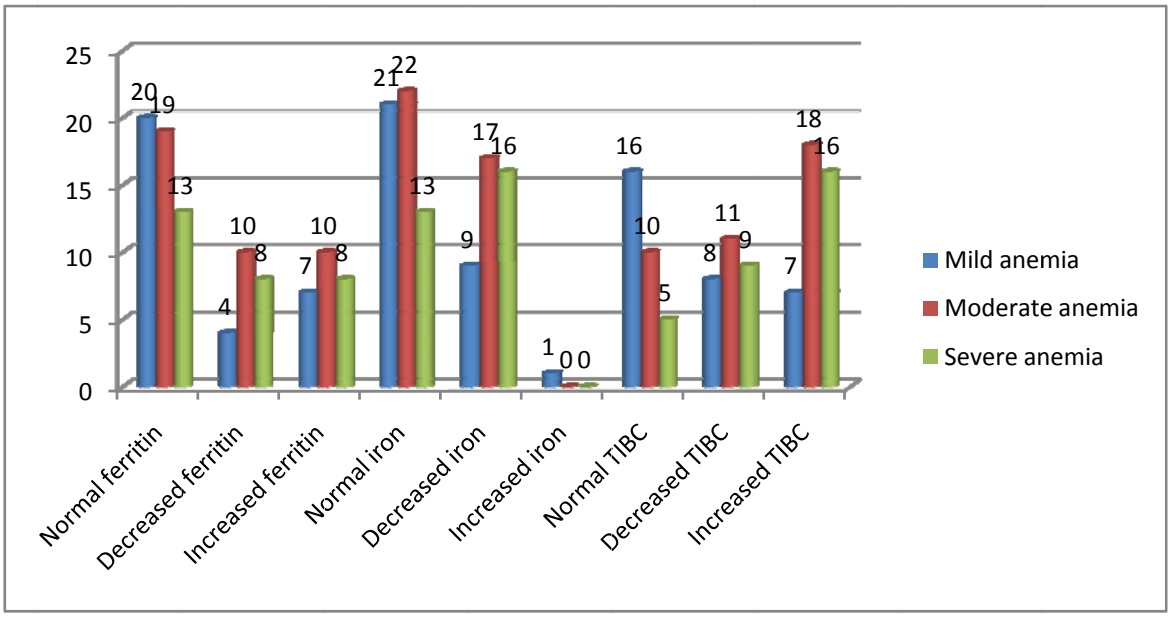
Iron levels were normal in 21, 22, 13 patients among the mild, moderate and severely anemic respectively. Low iron was seen in 9 mildly anemic, 17 moderately anemic and 16 severely anemic patients whereas only 1 patient in the mildly anemic group showed increased iron levels.

Total iron binding capacity was normal in 16 patients with mild anemia, decreased in 8 and increased in 7 of the same group. The level of TIBC in moderately anemic patients was normal in 10, decreased in 11 and increased in 18 patients. Among the severely anemic 5 had normal TIBC, 9 had decreased TIBC and 16 had increased TIBC.

TABLE NO. 19. COMPARISON BETWEEN IRON PARAMETERS AND INTENSITY OF ANEMIA

	Normal ferritin	Decreased ferritin	Increased ferritin	Normal iron	Decreased iron	Increased iron	Normal TIBC	Decreased TIBC	Increased TIBC
Mild anemia	20	4	7	21	9	1	16	8	7
Moderate anemia	19	10	10	22	17	0	10	11	18
Severe anemia	13	8	8	13	16	0	5	9	16

FIGURE NO.19. COMPARISON BETWEEN IRON PARAMETERS AND INTENSITY OF ANEMIA



CORRELATION OF PERIPHERAL SMEAR FINDINGS WITH INTENSITY OF ANEMIA

Normocytic normochromic erythrocytes is seen in a total of 50 patients out of which 19 were mildly anemic patients, 21 were moderately anemic patients and 10 were severely anemic patients. Microcytic hypochromic erythrocytes were found in a total of 44 patients of whom 10 were mildly anemic, 17 were moderately anemic and 18 were severely anemic patients. Dimorphic blood picture was seen totally 6 patients of whom 2 were mildly anemic, 1 was moderately anemic and 2 were severely anemic patients.

Normal WBC counts were seen in 21 mildly anemic, 25 moderately anemic and 22 severely anemic patients whereas leucopenia was seen 1 ,mildly anemic, 3 moderately anemic, 2 severely anemia and leucocytosis was seen 9 mildly anemic, 11moderately and 6 severely anemic patients.

Platelet counts were normal in 92 patients of whom 27 were mildly anemic, 38 were moderately anemic and 27 were severely anemic. Thrombocytopenia was present in 4 patients of which they were 2 each in mildly and severely anemic group. Thrombocytosis was seen in 3 patients of which 2 were mildly anemic and 1 patient was moderately anemic.

TABLE NO.20. ERYTHROCYTE MORPHOLOGY IN MILD, MODERATE AND SEVERE ANEMIA

	Normocytic normochromic (in %)	Microcytic hypochromic (in %)	Dimorphic (in %)
Mild anemia	19	9	3
Moderate anemia	21	17	1
Severe anemia	10	18	2
total	50	44	6

FIGURE NO.20 ERYTHROCYTE MORPHOLOGY IN MILD, MODERATE AND SEVERE ANEMIA

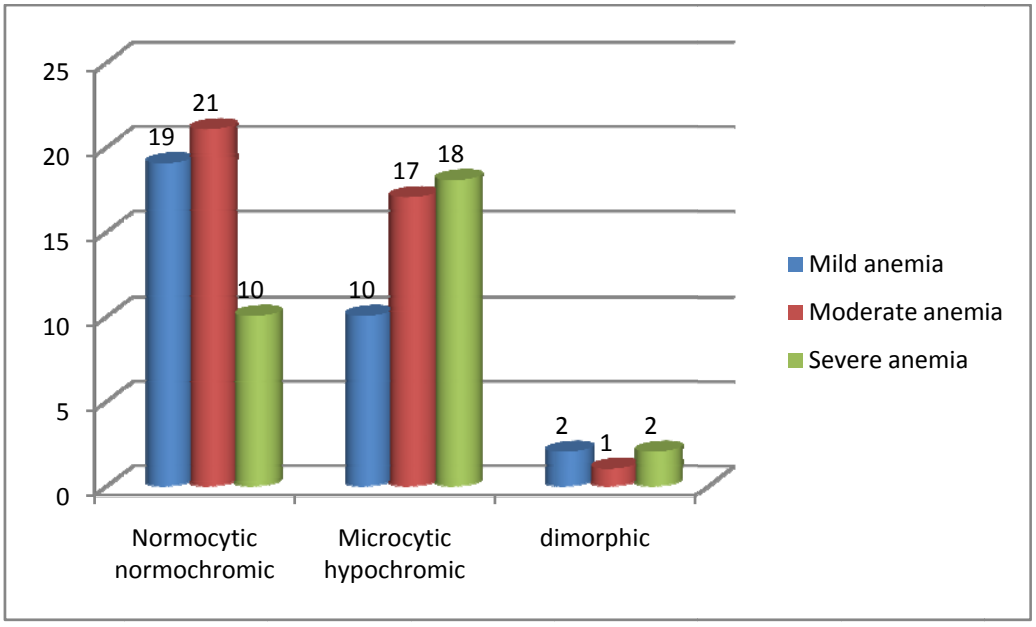


TABLE NO.21. WBC COUNTS IN MILD, MODERATE AND SEVERE ANEMIA

	Normal WBC count (in %)	leucocytosis (in %)	Leucopenia (in %)
Mild anemia	21	9	1
Moderate anemia	25	11	13
Severe anemia	22	6	2

FIGURE NO.21.WBC COUNTS IN MILD, MODERATE AND SEVERE ANEMIA

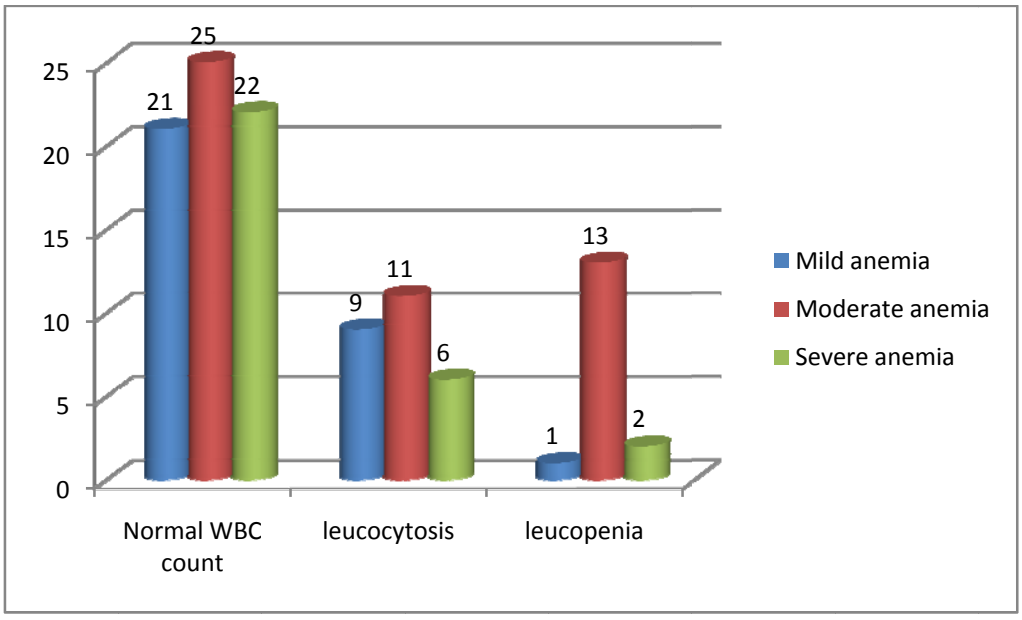
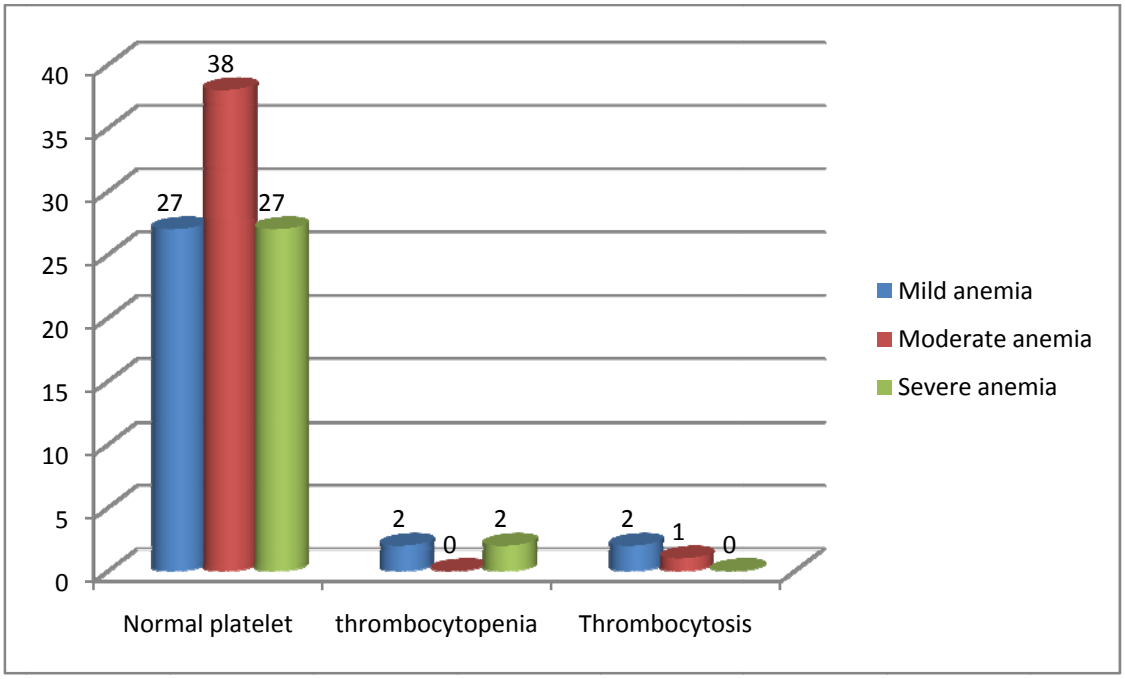


TABLE NO.22. PLATELET COUNTS IN MILD, MODERATE AND SEVERE ANEMIA

	Normal platelet (in %)	thrombocytopenia (in %)	Thrombocytosis (in %)
Mild anemia	27	2	2
Moderate anemia	38	0	1
Severe anemia	27	2	0

FIGURE NO.22. PLATELET COUNTS IN MILD, MODERATE AND SEVERE ANEMIA



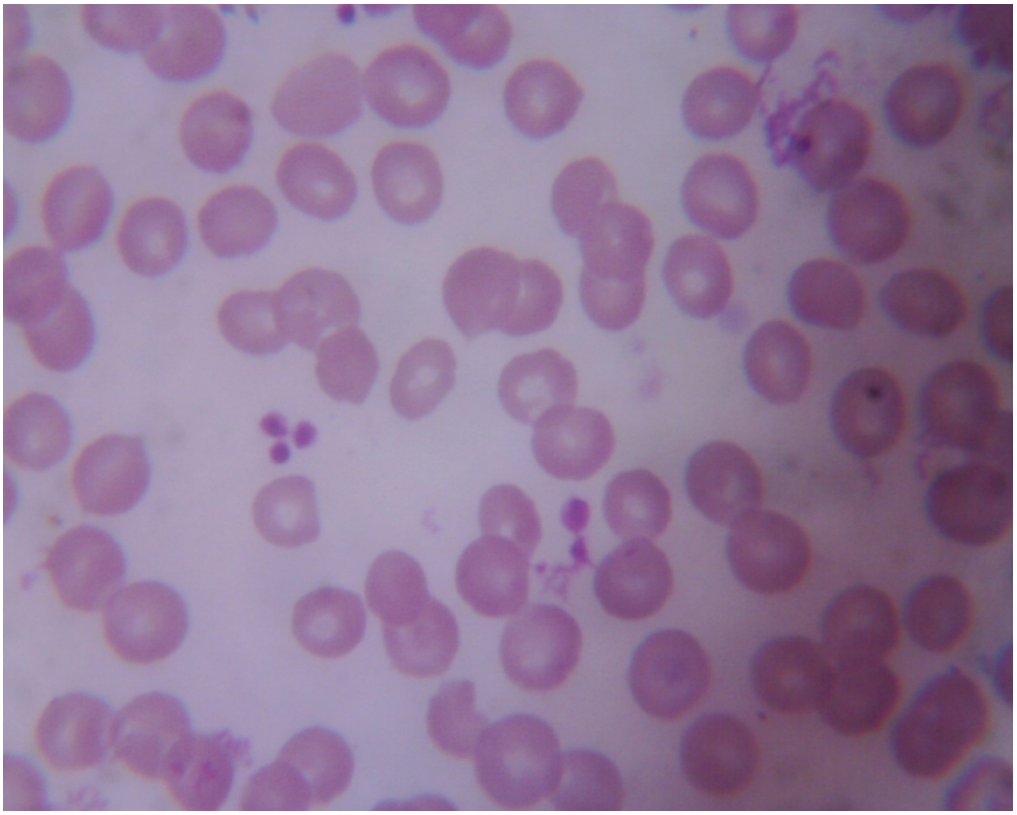


IMAGE 12. Photomicrograph showing peripheral smear with Normocytic Normochromic Red Blood Corpuscles (1000x, oil, leishman stain)

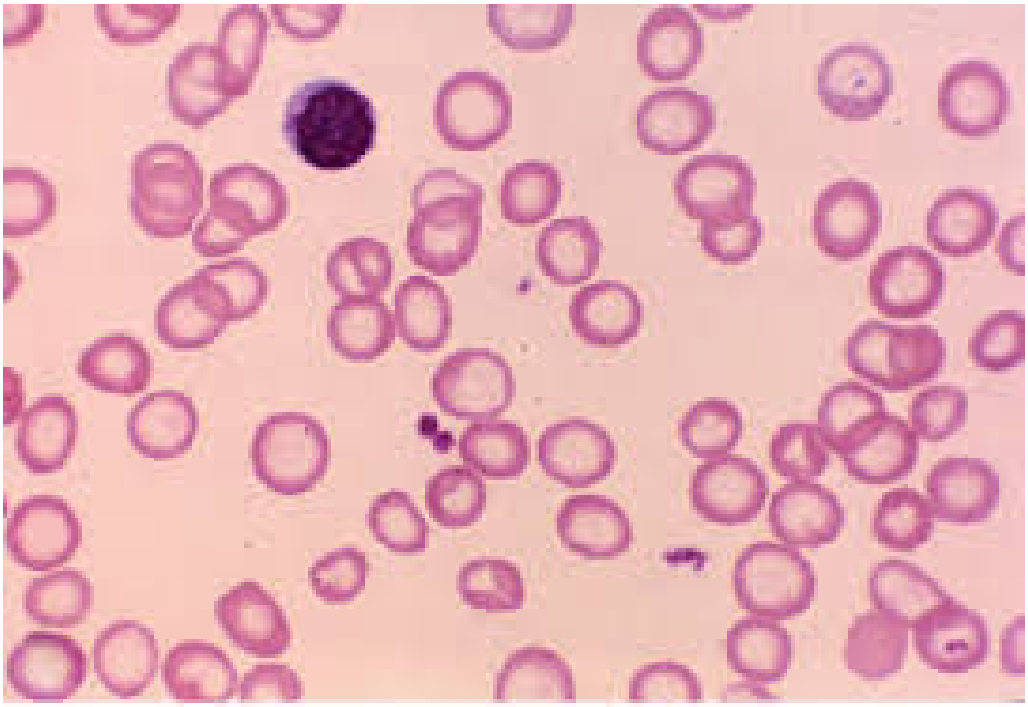


IMAGE 13. Photomicrograph showing peripheral smear with Microcytic Hypochromic Red Blood Corpuscles (1000x, oil, leishman stain)

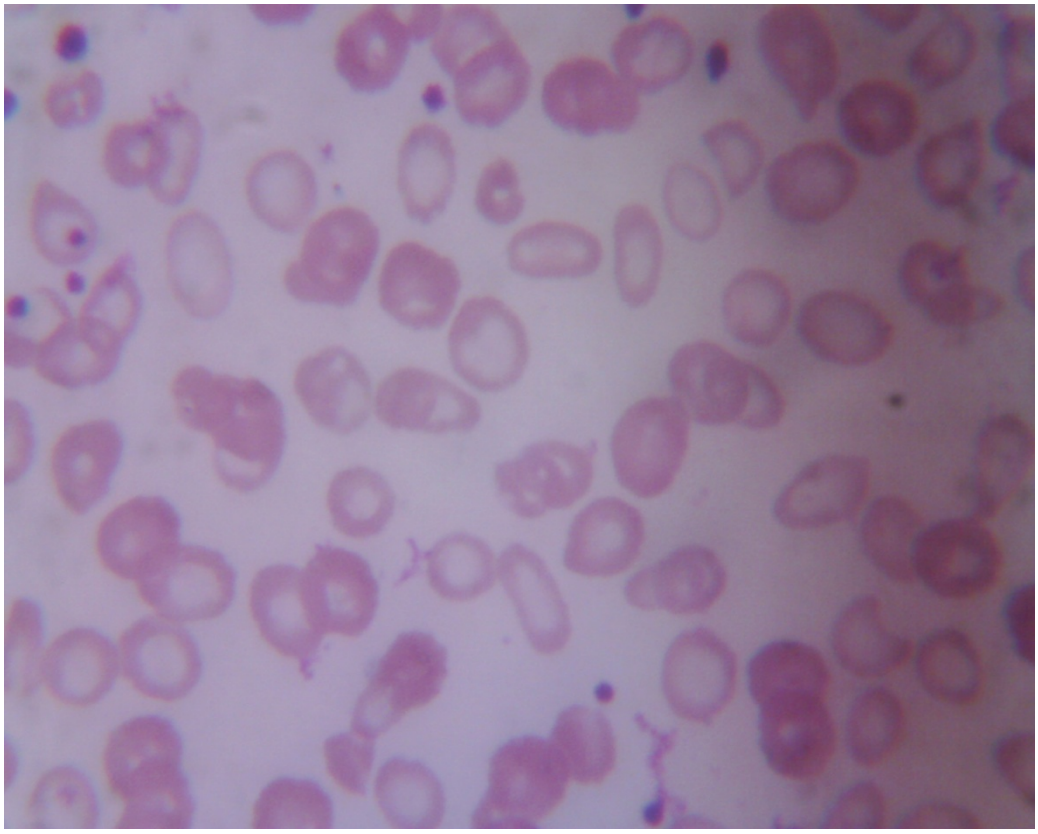


IMAGE 14. Photomicrograph showing peripheral smear with Dimorphic Blood Picture with Microcytic Hypochromic cells And Macrocytes (1000x, oil, leishman stain)

DISCUSSION

This study, conducted in a rural tertiary hospital, identified 100 old age persons with anemia (hemoglobin less than 13 gm/dl in men and less than 12 gm/dl in women) who were included in the study.

With regard to the various clinical features of anemia in old age in this study (table 7) the most common symptom was easy fatiguability which was seen in 75% of patients followed by peripheral edema seen in 10 % and palpitations in 9 % of patients. These features corroborate closely with the results of the study by Amit Bhasin et al 2010[138]. The clinical signs seen in this study was pallor in 92 %, peripheral edema in 21%, glossitis in 11%, koilonychia in 7%, hepatomegaly in 5 % and splenomegaly in 6%. All the clinical signs were of higher incidence than that seen by Amit Bhasin et al 2010[138] except peripheral edema which was similar in incidence.

Regarding the age, the age in the present study population (table 1) ranged from 65 to 96 years with the mean age being 70.38. This mean age is similar to the studies conducted by Amit Bhasin et al 2010[138] , Chul Won Choi et al 2003[139], slightly lower than that seen in study by Tettamanti M et al 2010[140] and Saurabh R Srivastava et al 2013[141]. In the present study the maximum number of patients were in the age group between 65 and 75 years comprising 85 % of the study population and this is similar to the study by Sfurti Mann et al 2014[142], Tettamanti M et al 2010[140] . The number of men (57 %) with anemia is higher than that of women (43 %) with anemia in the present study (table 2) and this similar to the studies conducted by Kaur et al 2013[143] and Chul Won Choi et al

2003[139], and different from those by Tettamanti M et al 2010[140] and Saurabh R Srivastava et al 2013[141], in which the percentage of women with anemia was found to be higher.

The examination of peripheral smears in this study showed normocytic normochromic anemia to be the commonest seen in 50% of the patients which is similar to the study by Sfurti Mann et al 2014[142], Kaur et al 2013[143] and lower than that seen in Tettamanti M et al 2010[140] and Chul Won Choi et al 2003[139], and higher than that seen by Saurabh R Srivastava et al 2013[141]. This is followed by microcytic hypochromic anemia which was found in 44% of patients which is slightly higher than that seen in the study by Sfurti Mann et al 2014[142], Kaur et al 2013[143]and significantly higher than that seen in study by Saurabh R Srivastava et al 2013[141], Tettamanti M et al 2010[140] and Chul Won Choi et al 2003[139] . Dimorphic anemia was seen in 6% of patients in this study which is slightly lower than that seen in study by Kaur et al 2013[143]and study by Sfurti Mann et al 2014[142] .

Table 23 : Comparison of peripheral smear findings in the present study with other studies

	PRESENT STUDY	KAUR ET AL 2013[143]	SFURTI MANN ET AL 2014[142]	SAURABH R SRIVASTAVA ET AL 2013[141].	TETTAMANTI M ET AL 2010[140]	CHUL WON CHOI ET AL 2003[139],
MCHC ANEMIA	44%	34%	40.40%	11.6%	72.3%	93.5%
NCHC ANEMIA	50%	56%	50%	69.8%	16.9%	3.5%
DIMORPHIC ANEMIA	6%	8%	10%	4.44%	-	-

Regarding the various etiologies (table 5) for anemia the commonest cause in the present study was iron deficiency anemia which differed from other studies like NHANES III[9], Sfurti Mann et al 2014[142], Kaur et al 2013[143] , Tettamanti M et al 2010[140] , Chul Won Choi et al 2003[139], which show anemia of chronic disease to be the commonest cause. The second most common cause for anemia in the present study was anemia of chronic disease followed by anemia due to chronic kidney disease and unexplained anemia.

Table 24 : Comparison of etiology of anaemia in the present study with other studies

	IDA	ACD	A-CKD	MEGALOBLASTIC ANEMIA	UNEXPLAINED ANEMIA
Present study	43%	32%	11%	1%	11%
NHANES III	14%	20%	8%	14%	34%
Sfurti Mann et al 2014[142],	35%	41.66%		0.3%	0.6%
Tettamanti M et al 2010[140]	16%	17.4%	15%	10.1%	26.4%
Chul Won Choi et al 2003[139],	7%	-	-	-	-

Regarding the iron parameters (table 15) in microcytic hypochromic anemia the ferritin values had a mean value of 61.75 µg/dl, in normocytic normochromic anemia it was 162.16 µg/dl and in dimorphic anemia it was 299 µg/dl which varied from other studies. Sfurti Mann et al 2014[142] showed that mean ferritin values in microcytic hypochromic anemia was 70.58 µg/dl, in normocytic normochromic anemia was 424.67µg/dl and in dimorphic anemia was 261.33µg/dl. Iron parameters in this study depending on etiology (table 16) showed the ferritin levels varying from that seen by Sfurti Mann et al 2014[142] as shown below.

Table 25 : Comparison of iron parameter in the present study with study by Sfurti Mann et al

	IDA Present study	IDA Sfurti Mann et al 2014[142],	ACD Present study	ACD Sfurti Mann et al 2014[142],
Ferritin µg/dl (mean)	23.58	17.38	220.51	504.2

Of the 43 patients with iron deficiency anemia only 32 patients had peripheral smear showing the characteristic microcytic hypochromic picture even though the iron studies showed values suggestive of iron deficiency. Of the 43 patients only 6 patients had history of GI bleed. Hence chronic blood loss could not be attributed to the iron deficiency and the deficiency is probably due to nutritional causes since almost all of the persons in the study population belong to the low socioeconomic status.

LIMITATIONS

- Many patients could not produce previous imaging studies and laboratory parameters
- B 12 and folate assay could not be done
- Patients with unidentified etiology could not be evaluated further

CONCLUSION

This study showed that the commonest cause for anemia among elderly patients is iron deficiency anemia followed by anemia due to chronic disease and also that it can be asymptomatic which is incidentally stumbled upon when one is evaluated for other symptoms. Not many clinical signs are consistent with anemia except for pallor even which can be absent in cases of mild anemia. Even though iron deficiency anemia is the commonest cause the peripheral smear studies in this study showed that normocytic normochromic picture was the commonest even when MCV levels were suggestive of microcytic anemia. Geriatric anemia is a disease that often goes unreported hence every effort should be made to identify the disease and evaluate the cause and it should not be ignored as merely being a part of ageing, for the consequences of anemia can have higher morbidity in the elderly.

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ANNEXURE I

PROFORMA

NAME

AGE

SEX

ADDRESS

OCCUPATION

DURATION AND DETAILS OF ILLNESS

SYMPTOMS

- Easy fatiguability
- Frequent falls
- Dizziness
- Tinnitus
- Palpitations
- Abdominal distention
- Symptoms of thyroid disturbances
- Recurrent infections

PAST HISTORY

- Diabetes
- Hypertension
- Coronary artery disease
- Tuberculosis
- Malignancies
- Chronic kidney disease

DRUG HISTORY

PERSONAL HISTORY

- Smoking
- Alcoholism

GENERAL EXAMINATION

- Pallor
- Icterus
- Pedal edema
- Lymphadenopathy
- Clubbing
- Koilonychia
- Glossitis
- Angular stomatitis
- Aphthous ulcers

SYSTEMIC EXAMINATION

CVS

RS

ABDOMEN

CNS

INVESTIGATIONS

- CBC
- ESR
- Stool for occult blood
- Blood urea
- Serum creatinine
- Liver function tests
- Peripheral smear
- Reticulocyte count
- Serum ferritin
- Serum iron
- Total iron binding capacity
- Imaging studies
- Endoscopic studies (if needed)

ஆராய்ச்சி தகவல் தாள்

திருநெல்வேலி மருத்துவ கல்லூரி அரசு பொதுமருத்துவமனைக்கு வரும் 65 வயதுக்கு மேற்பட்ட முதிய நோயாளிகளுக்கு ஏற்படும் இரத்தசோகை பற்றிய ஒரு ஆராய்ச்சி நடை பெற்றுவருகிறது.

முதிய நோயாளிகளுக்கு இரத்த சோகை என்பது எவ்வளவு பரவலாக இருக்கிறது என்பது பற்றியும், அதற்கான காரணங்கள் என்ன என்பது பற்றியும் அறிந்து கொள்வதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருத்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

<u>ஆராய்ச்சி ஒப்புதல் கடிதம்</u>

ஆராய்ச்சிதலைப்பு: 65 வயதுக்கு மேற்பட்ட முதிய நோயாளிகளுக்கு ஏற்படும் இரத்தசோகை பற்றிய ஆராய்ச்சி.

பெயர்: தேதி:

வயது: உள்நோயாளிஎண்:

பால்: ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இரத்தசோகை அறிகுறிகள் மற்றும் பாதிப்புகள் குறித்து ஆராய்ச்சியாளர் கூற முழுவதும் விளங்கப் பெற்றேன்.

இதற்குத் தேவையான உடற்பரிசோதனைக்கும், இரத்தம் சம்பந்தப்பட்ட பரிசோதனைகளுக்கும் மனமார சம்மதிக்கிறேன்.

கையொப்பம்

MASTER CHART

			CLINICAL FEATURES																								
S.NO	AGE	SEX	COMPLAINTS	EASY FATIGUABILITY	GI BLEED	ABDOMINAL DISTENTION	CHRONIC DRUG INTAKE	FALLS	SMOKING	ALCOHOLISM	DIABETES	HYPERTENSION	FEATURES OF RENAL DISEASE	STOOL FOR OCCULT BLOOD	THYROID SYMPTOMS	KNOWN MALIGNANCIES	PALPITATIONS	PALLOR	CLUBBING	ICTERUS	PEDAL EDEMA	KOILONYCHIA	GLOSSITIS	APHTHOUS ULCERS	LYMPHADENOPATHY	HEPATOMEGALY	SPLENOMEGALY	TACHYCARDIA
1	65	M	C/O ABDOMINAL DISTENTION	Y	N	Y	N	N	Y	Y	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	Y	Y
2	70	F	H/O FALLS,EASY FATIGUABILITY	Y	N	N	N	Y	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
3	65	M	EASY FATIG, JOINT PAINS	Y	N	N	Y	N	N	Y	N	N	N	ND	N	N	N	Y	N	N	N	Y	Y	N	N	N	N	N
4	69	M	COUGH,FEVER FOR 10 DAYS	Y	N	N	Y	N	Y	Y	Y	Y	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N
5	75	F	FEVER,DYSPNOEA,LOOSE STOOLS	Y	N	N	Y	N	N	N	Y	N	N	ND	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	N
6	80	M	ALTERED SENSORIUM,OLIGURIA	Y	N	Y	Y	N	Y	Y	Y	N	N	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
7	65	F	COUGH,FEVER FOR 10 DAYS	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	Y	Y	Y	N	N	N	N	N	N	N	N	Y
8	73	M	REVIEW	Y	N	N	Y	N	N	N	N	N	N	ND	N	Y	Y	Y	N	N	Y	N	N	N	Y	N	Y	N
9	67	F	PARAPARESIS	Y	N	N	Y	Y	N	N	N	Y	N	ND	N	N	Y	Y	N	N	N	Y	N	N	N	N	N	Y
10	70	F	REVIEW	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	N	Y	N	N	Y	N	N	N
11	68	F	FEVER, COUGH FOR 10 DAYS	Y	N	N	Y	N	N	N	Y	N	N	ND	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N
12	69	F	INGUINAL LYMPHADENOPATHY	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	Y	Y	N	N	Y	N	N	N
13	71	M	DYSPNOEA,COUGH EXPECTORATION	Y	N	N	Y	N	Y	Y	N	N	N	ND	N	N	Y	Y	Y	N	N	N	N	N	N	N	N	N
14	73	M	COUGH DYSPNOEA	Y	N	N	Y	N	N	N	Y	Y	N	ND	N	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N
15	69	M	DYSPNOEA,COUGH EXPECTORATION	N	N	Y	Y	N	Y	Y	N	N	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	Y	N	N
16	65	F	LUMP BREAST FOR 6 MONTHS	Y	N	N	N	N	N	N	Y	N	N	ND	N	Y	N	Y	N	N	N	Y	N	N	Y	N	N	N
17	74	F	LUMP BREAST FOR 3 YEARS	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N
18	96	M	GIDDINESS,	N	N	N	Y	N	N	N	Y	N	N	ND	N	N	N	Y	N	N	N	N	Y	Y	N	N	N	N
19	65	M	PEDAL EDEMA FOR 20 DAYS	Y	N	N	Y	N	Y	Y	Y	Y	Y	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
20	70	F	ACCIDENTAL FALL FRACTURE	Y	N	N	N	Y	N	N	N	N	N	ND	N	N	Y	Y	N	N	N	N	N	N	N	N	N	N
21	70	F	FEVER WITH CHILLS FOR 10 DAYS	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
22	80	M	SCROTAL SWELLING AND PAIN	N	N	N	Y	N	Y	Y	Y	N	N	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
23	70	M	COUGH EXPECTORATION FEVER 2 WKS	Y	N	N	Y	N	Y	Y	Y	Y	N	ND	N	Y	N	Y	Y	N	N	N	N	N	N	N	N	N
24	71	M	GIDDINESS ON AND OFF	Y	N	N	N	N	N	N	N	N	N	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
25	70	M	APHASIA, FOCAL SEIZURES	N	N	N	N	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
26	65	F	MASS PV THYROID LUMP	Y	N	N	N	N	N	N	N	N	N	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N	N
27	80	M	R HEMIPLEGIA	N	N	N	N	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
28	66	M	COUGH DYSPNOEA FEVER	N	N	N	N	N	Y	Y	Y	N	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N
29	67	F	NASAL MASS	N	N	N	Y	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
30	70	M	EASY FATIGUABILITY FEVER	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
31	65	M	PEDAL EDEMA ABD.DISTENTION	Y	N	Y	Y	N	N	N	Y	Y	Y	N	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N

32	67	F	THYROMEGALY L VOCAL CORD PALSY	N	N	N	N	N	N	N	N	N	N	ND	Y	N	N	Y	N	N	N	N	N	N	N	N	N
33	70	F	ACCIDENTAL FALL FRACTURE TIBIA	Y	N	N	N	N	N	N	N	N	N	ND	N	N	Y	Y	N	N	N	N	N	N	N	N	N
34	68	M	RTA HEMIATHROPLASTY 2 YRS BACK	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N
35	70	F	ACCIDENTAL FALL FRACTURE	Y	N	N	N	N	N	N	Y	Y	Y	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N
36	80	M	ASSAULT FRACTURE	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	N	N	N	N	N	N	N	N	N	N
37	65	M	LOC, APHASIA, DROOLING	Y	N	Y	Y	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N
38	80	M	GIDDINESS, TINNITUS	N	N	N	Y	N	N	N	Y	Y	Y	ND	N	N	N	N	N	N	N	N	N	N	N	N	N
39	65	M	GIDDINESS FALL HEMETEMESIS	Y	Y	Y	Y	Y	Y	Y	N	N	N	Y	N	N	N	Y	Y	Y	N	N	N	N	N	N	Y	N
40	67	F	SWELLING R SIDE JAW 1 MONTH	Y	N	Y	Y	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N
41	78	M	K/C/O CKD DYSPNOEA	Y	N	Y	Y	N	N	N	Y	Y	Y	ND	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N
42	67	F	FEVER DYSURIA	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	Y	N	Y	Y	N	N	N	N
43	73	F	ULCER R THIGH	Y	N	N	Y	N	Y	Y	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
44	70	M	ABDOMINAL PAIN VOMITTING	Y	Y	N	N	N	Y	Y	Y	Y	N	Y	N	Y	N	Y	N	N	N	Y	Y	Y	N	N	N	N
45	65	F	LUMP BREAST 4 MONTHS	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	N	N	N	N	N	N	N	Y	N	N	N
46	65	M	PAIN ABDOMEN VOMITING	Y	?	N	Y	N	Y	Y	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
47	75	M	FEVER, VOMITING,PAIN ABD	N	N	N	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
48	68	M	ACUTE CORONARY SYNDROME	Y	N	N	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
49	65	M	EASY FATIG, MALENA	Y	Y	Y	Y	Y	Y	Y	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
50	70	M	DYSPNOEA,COUGH EXPECTORATION	Y	N	N	Y	N	Y	Y	N	N	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N
51	70	M	ABDOMEN DISTENTION,	Y	N	Y	Y	N	Y	Y	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	Y	N	N
52	68	F	PEDAL EDEMA,DYSPNOEA	Y	Y	N	Y	N	N	N	Y	Y	Y	Y	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
53	65	F	GIDDINESS,DYSPNOEA	Y	N	Y	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	Y	Y	N
54	68	F	DYSPNOEA,K/C/O CAD	Y	N	Y	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
55	72	F	K/C/O DCLD	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	Y	N	N	Y	Y	Y
56	65	F	K/C/O CKD DYSPNOEA	Y	N	N	Y	N	N	N	N	N	Y	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
57	70	M	PARAPERESIS	Y	N	N	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	N
58	70	F	K/C/O CARCINOMA CERVIX	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	N	Y	Y	Y	N	N	N	N
59	72	M	K/C/O CKD ANASARCA	Y	N	Y	Y	N	N	N	Y	Y	Y	ND	N	N	Y	Y	N	N	Y	N	N	N	N	N	N	Y
60	70	F	COUGH FEVER DYSPNOEA	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y
61	68	M	EASY FATIGUABILITY	Y	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
62	76	F	FEVER DYSURIA	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	Y	N	N	N	N	Y
63	63	F	R SIDE HEMIPLEGIA	Y	N	N	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
64	70	F	EASY FATIGUABILITY, K/C/O AF	Y	N	N	Y	Y	N	N	N	N	N	ND	N	N	Y	Y	N	N	Y	N	N	N	N	N	N	Y
65	70	M	FEVER DIARRHOEA DYSURIA	Y	N	Y	N	N	N	Y	N	N	N	ND	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	Y
66	65	M	FEVER DYSPNOEA	Y	N	Y	Y	N	N	N	Y	Y	Y	ND	N	N	N	Y	N	N	Y	N	N	N	N	N	N	Y
67	70	M	K/C/O COPD	N	N	N	Y	N	Y	Y	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
68	75	M	VOMITING DYSPHAGIA	Y	Y	N	N	N	Y	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N	N	N	Y
69	65	M	FEVER, CLINICAL MALARIA	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
70	70	M	DYSPEPSIA	N	Y	N	Y	N	Y	Y	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
71	71	M	K/C/O PT RELAPSE	Y	N	N	Y	N	Y	Y	N	N	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N
72	70	F	K/C/O CARCINOMA OVARY	Y	N	Y	N	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	Y	N	N	Y	N	N	N	Y
73	68	F	FEVER COUGH NEW PT	N	N	N	N	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
74	69	F	K/C/O PT COMPLETED ATT	Y	N	N	Y	N	N	N	N	N	N	ND	N	N	N	Y	Y	N	N	N	Y	N	N	N	N	N

75	65	F	FEVER DYSURIA	N	N	N	N	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N
76	68	F	RLOIN PAIN DYSURIA	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
77	75	M	URINARY RETENTION POLYURIA	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
78	76	M	K/C/O CKD	Y	N	Y	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
79	80	M	FALL INTERTROCHANTERIC FRAC	N	N	N	N	Y	N	N	N	N	N	ND	N	N	N	N	N	N	N	N	N	N	N	N	N	N
80	70	M	HEMETEMESIS,MALENA	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
81	75	M	EPISTAXIS	N	N	N	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
82	78	M	R HYPOCHONDRIAC PAIN FEVER	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	N	N	N	N	N	N	N	N	N	N	N
83	65	F	GIDDINESS FALL HEMIPARESIS	Y	N	N	Y	N	N	N	Y	Y	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
84	65	M	DYSPNOEA COUGH	Y	N	N	N	N	Y	Y	N	N	N	ND	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N
85	74	M	K/C/O DCLD HEMETEMESIS	Y	Y	Y	N	N	Y	Y	Y	Y	N	Y	N	N	N	Y	N	Y	N	N	N	N	N	N	Y	Y
86	72	F	BEHAVIOURAL DISTURBANCES	N	N	N	N	Y	N	N	N	N	N	ND	N	N	N	N	N	N	N	N	N	N	N	N	N	N
87	65	F	ABDOMEN PAIN VOMITING	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N
88	65	F	GIDDINESS	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
89	65	F	K/C/O PEMPHIGUS VULGARIS	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
90	74	M	INGUINAL LYMPHADENOPATHY	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
91	65	M	RECURRENT FEVER	Y	N	N	N	N	Y	Y	N	N	N	ND	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N
92	80	M	FALL INTERTROCHANTERIC FRAC	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
93	72	M	K/C/O CARCINOMA RECTUM	Y	Y	N	N	N	Y	Y	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N
94	70	M	ETHMOIDAL POLYP OPERATED	N	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
95	66	F	FEVER DIARRHOEA	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N
96	76	F	FUNGATING MASS R BREAST	Y	N	N	N	N	N	N	N	N	N	ND	N	Y	N	Y	N	N	N	N	N	N	Y	N	N	N
97	87	M	GIDDINESS TINNITUS	Y	N	N	N	N	N	N	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y
98	70	M	PEDAL EDEMA OLIGURIA	Y	N	Y	N	N	Y	Y	N	N	N	ND	N	N	N	Y	N	Y	Y	N	N	N	N	N	N	N
99	72	M	DYSPNOEA	Y	N	N	N	N	Y	Y	N	N	N	ND	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	N
100	67	F	CHEST DISCOMFORT	Y	N	N	N	N	Y	Y	N	N	N	ND	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y

LABORATORY PARAMETERS																	
S.NO	**HEMOGLOBIN**	**RBC COUNT**	**WBC TOTAL COUNT**	**DIFFERENTIAL COUNT**	**PCV**	**PLATELET COUNT**	**ESR**	**MCV**	**MCH**	**MCHC**	**RDW**	**RPI**	**FERRITIN**	**SERUM IRON**	**TIBC**	**RENAL PARAMETERS**	**LIVER FUNCTION TESTS**	**ULTRASONOGRAM**
1	12	7.14	23800	78/13/2	39	5.73	10	64	18	23	13.2	2.6	46.9	30	346	NORMAL	NORMAL	SPLENOMEGALY WITH NORMAL PORTAL VEIN THICKNESS
2	5.8	3.9	5200	66/25/7	23	3.74	142	54	16	28	23.2	0.4	50.3	14	542	NORMAL	NORMAL	NORMAL STUDY
3	6.7	3.7	4700	64/31/2	34	1.46	115	72	18.1	25	20.2	0.8	3	10	633	NORMAL	NORMAL	NORMAL STUDY
4	10.7	3.37	4900	65/20/15	30.2	1.28	105	90.8	31.8	33.2	14.2	1.1	360	60	372.8	ELEVATED	NORMAL	CHRONIC KIDNEY DISEASE,PROSTATOMEGALY
5	10.1	3.82	20500	90/8/5	29.9	7300	132	76	25.8	33.8	16.6	1	86	16.88	55.32	22/1.1	NORMAL	NORMAL STUDY
6	6.6	2.73	4300	69/20/11	21.8	1.11	40	79.9	24.2	30.3	13.3	1.2	330.83	37.25	177.03	22/1.1	NORMAL	PARENCHYMAL LIVER DISEASE WITH PHT,

7	9.7	3.66	15200	80/5/15	23	2.73	44	80.3	26.5	33	14.2	0.6	223.3	56.2	123.9	NORMAL	NORMAL	NORMAL STUDY
8	10	3.44	43000	30/68/2	31	1.35	23	90	31.7	35.2	13.9	1.8	341.9	43.2	221.1	NORMAL	NORMAL	SPLENOMEGALY
9	5.1	3.42	11900	61/31/7	20	1.3	50	53.6	13.6	25.6	24.7	0.82	25.73	131	325.9	NORMAL	NORMAL	NORMAL STUDY
10	10	3.3	6200	64/27/9	30.4	2.83	30	92.1	30.9	33.6	13.3	0.5	86.9	65.87	445.8	NORMAL	NORMAL	NORMAL STUDY
11	6.6	2.05	2600	34/55/11	18.9	6000	40	92	32.2	34.9	20.2	1.01	225	84.05	117.36	NORMAL	NORMAL	NORMAL STUDY
12	10.3	3.87	9200	65/28/7	34.8	2.64	10	88.1	30.2	34.2	12.7	1.49	273.8	58.96	120	NORMAL	NORMAL	NORMAL STUDY
13	12	5.03	13200	76/20/4	39.4	2.22	15	78.3	23.9	30.5	15.3	0.2	36.66	50.6	168.63	NORMAL	NORMAL	NORMAL STUDY
14	11.4	3.96	8500	68/18/14	34.2	3.08	40	86.4	28.8	33.3	18.2	0.86	296.6	27.09	72.48	NORMAL	NORMAL	NORMAL STUDY
15	4.2	2.1	8500	74/17/9	17.5	4.8	110	83.5	20	24	20.1	0.44	27.43	57.51	138	NORMAL	NORMAL	HEPATOMEGALY
16	10.1	6.3	7700	48/47/5	55.8	1.53	10	87.5	26.8	30.6	14.9	0.67	149.77	63.79	59.85	NORMAL	NORMAL	NORMAL STUDY
17	10.1	3.82	6400	67/26/7	31.3	3.52	115	81.9	26.4	32.2	15.2	2.06	51.22	57.35	179.07	NORMAL	NORMAL	NOT DONE
18	8.1	3.76	8900	74/16/10	28.8	2.34	60	76.5	24.7	32.3	17.4	1.4	18.45	24.83	549.67	51/1.8	NORMAL	CHOLELITHIASIS
19	9	3.29	8000	66/24/10	25.7	2.92	40	78.1	27.4	35	14.4	0.2	282.3	88	131.52	NORMAL	NORMAL	GALL BLADDER CALCULI
20	6.8	3.3	6500	73/20/7	20	1.65	60	70.3	26.6	32	17.8	1.02	18.73	9.14	507.22	NORMAL	NORMAL	NORMAL STUDY
21	7.1	3.25	14500	88/8/3	24	4.7	90	73.8	21.8	29.6	20.6	1.05	21.2	19.84	462.19	NORMAL	NORMAL	SPLENIC ABCESS
22	11.3	3.51	9500	83/11/5	34.4	3.53	25	98	32.2	32.8	14.9	1.7	12.62	37.04	122.97	NORMAL	NORMAL	MINIMAL FLUID IN LOWER ABDOMEN
23	10.8	4.72	8000	80/9/9	33.2	5.11	28	70.3	22.9	32.5	13.4	1.7	191	70.23	142.59	NORMAL	NORMAL	NOT DONE
24	11	4.12	7000	64/26/8	38.5	2.46	15	93.4	29.1	31.2	13.8	0.18	385.7	28.52	324.9	NORMAL	NORMAL	NOT DONE
25	10.3	3.54	7400	77/13/8	30.2	2.8	15	85.3	29.1	34.1	12.6	0.93	57.05	21.92	355.2	NORMAL	NORMAL	NOT DONE
26	7.9	4.37	5800	65/23/11	28	2.5	10	65.7	18.1	27.5	28.6	1.3	4.86	16.16	588.9	NORMAL	NORMAL	NORMAL STUDY
27	8.7	3.5	6700	74/16/9	29.4	2.25	45	81.9	24.2	29.6	16.8	0.63	28.4	68.57	96.48	NORMAL	NORMAL	NORMAL STUDY
28	9.7	3.5	13300	85/7/6	25.3	1.78	20	79.2	27.6	34.9	12.4	1.36	223.1	48.4	266.64	NORMAL	NORMAL	NOT DONE
29	11	4.6	6800	67/25/6	29	3.44	2	38.8	26.6	38.8	13.7	0.84	18.1	48.07	456.9	NORMAL	NORMAL	NOT DONE
30	5.6	2.39	6000	76/12/10	24.5	1.43	30	77.8	23.4	30.1	24	0.43	5.34	16.16	530.8	NORMAL	NORMAL	NORMAL STUDY
31	9.4	3.21	14500	70/27/2	27.8	2.91	30	86.6	29.3	33.8	14.7	1.71	41.3	15.64	151.75	80/2.1	NORMAL	MRD GR I, PROSTATOMEGALY
32	11	4.02	9800	50/36/14	33	2.5	20	84.3	28.9	33.9	12.8	1.9	60.2	79.22	282.32	NORMAL	NORMAL	NOT DONE
33	6.3	2.2	5400	60/30/9	19.8	3.84	65	90	28.6	31.8	16.8	0.68	53.89	85.04	343.5	NORMAL	NORMAL	NOT DONE
34	8.4	2.98	6900	78/11/10	25.1	1.56	45	84.2	28.2	33.5	13.7	1.34	6.55	5.09	551.5	NORMAL	NORMAL	NORMAL STUDY
35	8.9	3.28	5000	58/29/2	27.1	2.51	20	82.6	27.1	32.8	13.8	0.78	50.74	60.31	160.3	62/1.5	NORMAL	GALL BLADDER CALCULI
36	12	3.87	5300	64/26/9	37	2.39	20	95.6	33.9	35.4	13.3	2.3	48.6	37.3	351.3	NORMAL	NORMAL	NORMAL STUDY
37	6.6	3.7	3700	66/24/9	23.6	9500	43	70	19.6	28	22	1.2	46.37	28.91	523	NORMAL	NORMAL	NORMAL STUDY
38	12	4.64	15000	77/15/7	39.9	2.27	2	86	31.9	37	13.5	2.1	63.12	18.49	424.8	NORMAL	NORMAL	MRD GRADE I, PROSTATOMEGALY
39	8.1	2.58	6100	53/23/23	23.1	1.28	10	89.5	31.4	35.1	16.2	0.83	10.68	54.08	360.9	NORMAL	ELEVATED	SPLENOMEGALY, DCLD WITH PHT
40	11	4.22	5100	56/31/10	35.4	2.1	2	83.9	28.7	34.2	12.8	1.9	40.42	197.3	218.49	NORMAL	NORMAL	NORMAL STUDY
41	8.9	3.17	9200	71/28/	26.5	2.23	25	83.6	28.1	33.6	15.4	1.12	12.38	8.57	552.3	160/5.7	NORMAL	MRD GRADE IV
42	7	2.55	7700	78/15/7	21.4	1.97	140	83.9	27.5	32.7	14.9	0.93	124.1	60.4	68.73	105/3.6	NORMAL	GRADE IV MRD
43	9.9	3.78	6300	75/17/8	32.3	2.01	150	85.4	26.2	30.7	19.4	0.72	61.9	18.81	331.8	46/1.4	NORMAL	GRADE I MRD
44	8.9	3.54	6700	75/17/8	30.2	1.52	110	82.3	25.2	30.7	16.2	0.85	255.8	32.3	402.7	43/1.3	NORMAL	ANTRAL WALL THICKENING
45	11	4.93	7600	71/22/6	40.7	2.67	22	82.6	28.8	34.9	13.1	1.8	615.9	34.96	352.2	NORMAL	NORMAL	NOT DONE
46	6.6	4.2	4600	60/18/22	21.9	1.08	42	79.9	24.1	30.1	24.1	0.82	251.1	22.77	73.56	NORMAL	NORMAL	NORMAL STUDY
47	9.4	3.65	3400	85/10/5	28.1	2.62	45	77	25.8	33.5	15	0.38	31.07	54.3	431.74	65/1.2	NORMAL	NORMAL STUDY
48	6.4	4.07	5900	76/16/7	24.4	4.83	35	59.5	15.7	26.4	19.3	0.77	28.84	17.45	509.41	NORMAL	NORMAL	CHOLELITHIASIS
49	4.6	2.83	2400	62/26/10	18.4	4.25	15	65	16.3	25	21.3	0.31	13.3	45.9	678	NORMAL	NORMAL	NORMAL STUDY

50	11	4.83	9800	83/13/1	39.1	3.6	15	81	26.3	32.5	14	0.93	53.14	58.2	382.3	NORMAL	NORMAL	NORMAL STUDY
51	7.4	3.91	2400	59/28/12	26.9	1.06	10	68.8	18.9	27.5	21	1.3	28.9	27.38	486.9	NORMAL	NORMAL	IVF DILATED
52	6.8	3.03	6800	59/29/11	21.7	3.75	20	71.6	22.4	31.3	20.5	1.2	50.25	9.09	427.5	48/1.6	NORMAL	NORMAL STUDY
53	7.1	2.59	2400	27/70/3	21.9	1.79	120	84.6	27.4	32.4	19.7	0.94	19.9	26.2	542.2	NORMAL	NORMAL	HEPATOSPLENOMEGALY
54	7	2.57	2500	62/30/8	21.8	1.71	20	84.8	27.2	32.1	19.9	0.44	42.48	15.9	253.04	NORMAL	NORMAL	NORMAL STUDY
55	9.6	3.35	4700	60/21/7	28.5	5.8	10	85.1	28.7	33.7	14.7	1.21	10.2	80.8	450.4	NORMAL	ELEVATED	HEPATOSPLENOMEGALY
56	9.7	3.35	10600	69/19/11	26.5	3.05	20	79.1	29	36.6	12.9	0.63	142.8	82.9	80.58	50/2.9	ELEVATED	GRADE II RENAL PARENCHYMAL DISEASE
57	6.8	2.28	7000	87/9/3	19.3	2.25	115	84.6	29.8	35.2	16	0.93	56.6	68.9	356.9	107/2	NORMAL	FATTY LIVER, RPD
58	8.8	4.37	8300	72/16/12	29.7	3.74	44	76.3	26.2	27.3	16.6	0.73	178.9	32.9	156.9	NORMAL	NORMAL	NORMAL STUDY
59	3.8	2.06	7500	75/15/10	13.8	3.93	54	67	18.4	27.5	15.5	0.43	332.2	24.9	131.9	115/2.3	NORMAL	GRADE IV MEDICAL RENAL DISEASE, ASCITES
60	6.8	3.57	7500	89/13/7	23	3.16	105	64.4	19	29.6	20.2	1.2	13.1	17.9	668.4	NORMAL	NORMAL	NORMAL STUDY
61	6.1	3.8	6900	73/24/3	22.3	2.26	56	78.3	27.3	27	26.3	1.8	24	58.9	480.9	NORMAL	NORMAL	PROSTATOMEGALY
62	4.3	2.01	4800	55/28/17	14.9	2.96	32	74	21	28	28.4	2.1	19.5	41.3	567.8	NORMAL	NORMAL	NORMAL STUDY
63	7.3	4.2	6500	68/20/12	28.3	1.68	30	80.6	28.3	37	13.3	3.4	179	26.6	187	NORMAL	NORMAL	NOT DONE
64	2.4	2.46	6700	73/24/3	13.3	1.8	108	61	16.7	27.3	20.6	0.34	20.9	43.6	654.3	NORMAL	NORMAL	NOT DONE
65	6.7	2.03	10600	89/6/4	14.2	76000	56	108	33	35.8	23	1.1	299	87.9	176.8	58/2	NORMAL	GRADE I RPD
66	10.8	3.98	15500	87/6/6	26.9	2.13	45	72.9	27.1	37.2	13.3	2.1	278.5	58.9	203.8	173/3.8	NORMAL	GRADE IV MEDICAL RENAL DISEASE, ASCITES
67	9.6	2.8	6700	54/28/16	22.4	1.71	33	91.3	33.3	36.5	12.7	2.1	343.9	43.2	176.9	NORMAL	NORMAL	NOT DONE
68	5.7	1.86	6100	70/20/9	15	1.92	106	87.1	30.6	35.2	13.3	1.2	543.5	33.2	123.2	NORMAL	NORMAL	NORMAL STUDY
69	10.8	3.4	9400	76/14/9	29	2.4	38	83.8	31.2	37.2	12.9	2.2	45.4	78.2	243.8	NORMAL	NORMAL	NORMAL STUDY
70	9.5	3.9	9200	78/13/7	28.9	3.96	45	81.7	29.1	35.6	15.5	1.8	299.9	76.9	387.9	NORMAL	NORMAL	NORMAL STUDY
71	8.6	3.8	13400	78/12/10	25.6	2.23	58	85.6	28.6	32.7	14.4	1.5	376	49.6	298.9	NORMAL	NORMAL	NORMAL STUDY
72	7.6	2.3	6800	80/10/10	15.9	1.45	82	68.3	24.2	28.3	18.9	0.56	20.8	43.2	567.1	NORMAL	NORMAL	MINIMAL ASCITES
73	10	3.6	12000	57/35/8	22.8	2.2	92	79	29.7	30.4	14.8	1.1	34.2	56.1	354.9	NORMAL	NORMAL	NORMAL STUDY
74	9.3	4.3	14000	68/20/12	28.4	3.25	54	74.3	28.3	33.3	16.38	1.3	16.7	54.2	455.1	NORMAL	NORMAL	NORMAL STUDY
75	10.5	3.8	10600	75/20/5	26.9	1.68	20	63.3	20.6	28.3	23.6	0.54	43.2	55.3	234.3	NORMAL	NORMAL	NORMAL STUDY
76	10.5	4.1	11000	70/20/10	27.8	2.25	23	75.5	23.6	29.4	16.2	0.65	9.87	11.2	487.9	NORMAL	NORMAL	LEFT LOWER URETERIC CALCULUS
77	9.6	3.63	6500	58/30/12	25	3.63	32	74	28.7	32.3	18.8	0.89	27.6	57.2	434.2	NORMAL	NORMAL	PROSTATOMEGALY
78	9.2	4.63	7000	68/14/18	28.3	1.86	40	89.4	29.6	32.4	14.6	1.2	245.6	30.4	176.2	67/2.6	NORMAL	GRADE IV MEDICAL RENAL DISEASE, ASCITES
79	10.3	3.84	8500	75/20/5	39.3	1.68	68	63.2	26.4	33.2	22.1	2.1	25.7	33.4	456.1	NORMAL	NORMAL	NORMAL STUDY
80	8.9	3.86	12000	65/30/5	16	5.68	50	88.3	29.4	35.4	12.2	1.5	27.7	54.4	451.1	NORMAL	NORMAL	NORMAL STUDY
81	11	4.83	9000	68/30/2	29.2	5.8	20	92.3	30.4	37.3	14.3	0.82	45.2	88.5	364.9	NORMAL	NORMAL	NORMAL STUDY
82	10.5	3.82	6700	70/20/10	35.3	1.33	25	70.3	26.4	32.3	17.8	1.9	23.2	54.2	545.2	NORMAL	NORMAL	CHOLELITHIASIS
83	5.3	2.23	6500	80/10/10	19.2	2.64	60	53.2	18.4	23.2	28.9	0.32	10.9	23.2	672.1	NORMAL	NORMAL	NORMAL STUDY
84	6.8	3.3	13400	82/10/8	25	1.64	60	86	26.3	30.3	16.2	0.63	555.2	78.3	122.1	NORMAL	NORMAL	NORMAL STUDY
85	6.4	3.8	7600	81/17/2	22.3	4.68	70	75.6	27.4	32.4	18.8	0.72	23.4	22.2	325.9	NORMAL	ELEVATED	SPLENOMEGALY WITH PHT
86	10.4	3.61	6500	69/23/8	28.2	1.8	30	82.5	28.8	34.9	15.4	1.8	12.9	13.2	455.2	NORMAL	NORMAL	NOT DONE
87	9.7	3.38	9200	68/27/4	26	1.09	32	85.8	28.7	33.4	16.2	1.2	34.3	43.2	234.1	NORMAL	NORMAL	HEPATOMEGALY,CHOLEDOCHOLITHIASIS
88	8.5	3.18	8800	60/31/9	20	3.5	40	77	26.7	34.7	19.8	0.87	12.9	23.2	433.4	77/2.5	NORMAL	NORMAL STUDY
89	5.4	1.97	10000	70/20/10	15.6	2.5	56	65.8	27.4	34.6	15.6	0.23	16.2	19.6	545.9	NORMAL	NORMAL	NOT DONE
90	7.9	3.48	16500	80/15/5	28.3	1.82	82	81.4	25.2	33	19.3	1.1	556.4	96.9	234.2	NORMAL	NORMAL	NORMAL STUDY

91	3.3	1.28	28600	23%BLAST	18.3	1.2	86.2	28.2	35	90	18.3	0.68	243.2	34.2	234.1	NORMAL	NORMAL	HEPATOMEGALY
92	8.8	3.2	9800	72/25/5	28	1.3	18	89.1	29.3	34.8	14.2	0.93	78.2	56.9	342.2	NORMAL	NORMAL	NORMAL STUDY
93	7.5	3.7	15000	73/24/3	21	2.26	82	88.3	29.2	33.4	13.7	1.3	333.4	56.6	123.4	NORMAL	NORMAL	PARAAORTIC LYMPH NODES ENLARGED
94	10	3.9	5800	75/20/5	29.7	2.28	33	76.4	24.2	30.1	19.7	1.6	37.7	89.2	342.9	NORMAL	NORMAL	NORMAL STUDY
95	9.8	3.2	11500	80/15/5	19.6	1.78	43	65.2	22.1	28.2	23.9	0.87	13.6	25.8	478.9	NORMAL	NORMAL	NORMAL STUDY
96	6.8	2.9	6900	83/10/7	23.9	2.89	98	89	28	32	15.6	0.9	349.5	66.8	167.2	NORMAL	NORMAL	NORMAL STUDY
97	6.7	3.3	5400	65/32/3	22	1.9	43	65	21	23	21.8	1.1	24.2	33.2	453.9	NORMAL	NORMAL	NORMAL STUDY
98	7.9	2.76	6000	59/31/11	24.6	1.84	37	87.4	25.9	33.7	13.7	1.6	23.2	34.3	439.2	NORMAL	ELEVATED	DCLD ASCITES
99	4.5	2.5	6000	67/22/11	16	1.99	62	80.3	26.9	31	14.9	0.98	33.2	54.2	527.6	NORMAL	NORMAL	ASCITES WITH FATTY LIVER
100	7.4	2.8	7800	67/30/3	22.8	2.3	62	68.9	23.9	29.8	19.9	1.1	23.9	43.3	458.9	NORMAL	NORMAL	NORMAL STUDY

S.NO	PERIPHERAL SMEAR	CHEST XRAY	CT SCAN	OTHER INVESTIGATIONS	DIAGNOSIS
1	NCNC MODERATE ANISOPOIKILOCYTOSIS,WBC NORMAL, EOSINOPHILIA, THROMBOCYTOSIS	NORMAL	NORMAL	SERUM ELECTROPHORESIS- B THALASSEMIA TRAIT	B THALASSEMIA TRAIT
2	MCHC,MOD AIP, MACROCYTES,STOMATOCYTES,ELLIPTOCYTES,WBC PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
3	NCNC,MILD ROULEAUX,ELLIPTOCYTES,TRANSFORMED MONOCYTES, PLATELETS ADEQUATE	NOT AVAILABLE	NOT DONE	-	IRON DEFICIENCY ANEMIA
4	NCNC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
5	NCHC ANEMIA NEUTROPHILIA THROMBOCYTOPENIA	NOT DONE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
6	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	COSTOPHRENIC ANGLE OBLITERATED	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
7	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	MEDIASTINAL MASS	MEDIASTINAL MASS	BIOPSY OF MASS- THYMOMA TYPE B1	ANEMIA OF CHRONIC DISEASE
8	MICROCYTIC HYPOCHROMIC ANEMIA,CHRONIC MYELOID LEUKEMIA, THROMBOCYTOPENIA	NOT AVAILABLE	NOT DONE	-	CHRONIC MYELOID LEUKEMIA
9	SEVERE MCHC ANEMIA, WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
10	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT AVAILABLE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
11	MICROCYTIC HYPOCHROMIC RBCS ADMIXED WITH MACROCYTES,LEUCOPENIA,THROMBOCYTOPENIA	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
12	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	FNAC INGUINAL NODE- MALIGNANT MELANOMA METASTATIC DEPOSITS	ANEMIA OF CHRONIC DISEASE
13	MICROCYTIC HYPOCHROMIC RBCS WITH MILD ANISOPOIKILOCYTOSIS,NL, PLATELETS ADEQUATE	R UPPER LOBE CALCIFICATION	MASS R UL WITH CALCIFICATION	FNAC- INFLAMMATORY SMEAR, BIOPSY MOD.DIFF SQUAMOUS	ANEMIA OF CHRONIC DISEASE

				CELL CARCINOMA	
14	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	R UPPER LOBE OPACITY SEEN	R UL MASS	BRONCHOSCOPY- R UL MASS R MAIN BRONCHUS 2 CM DISTAL TO CARINA	ANEMIA OF CHRONIC DISEASE
15	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	THICKWALLED CAVITY LEFT LUNG APICAL REGION	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
16	NORMOCYTIC NORMOCHROMIC RBCS LYMPHOCYTOSIS, PLATELETS NORMAL	NOT DONE	NOT DONE	BIOPSY AXILLARY NODE- METASTATIC DEPOSITS	ANEMIA OF CHRONIC DISEASE
17	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	FNAC BREAST MASS- DUCTAL CARCINOMA GRADE III,,NODE- METASTATIC CARCINOMATOUS DEPOSITS	ANEMIA OF CHRONIC DISEASE
18	MICROCYTIC HYPOCHROMIC RBCS WITH MILD ANISOPOIKILOCYTOSIS,NL, PLATELETS ADEQUATE	CARDIOMEGALY	NOT DONE	-	IRON DEFICIENCY ANEMIA
19	NORMOCYTIC NORMCOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	FUNDOSCOPY- MILD NON PROLIFERATIVE DIABETIC RETINOPATHY	ANEMIA OF CHRONIC DISEASE
20	MICROCYTIC HYPOCHROMIC ANEMIA, WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
21	SEVERE MCHC ANEMIA, NEUTROPHILIA, PLATELETS ADEQUATE	NOT AVAILABLE	NOT DONE	-	IRON DEFICIENCY ANEMIA
22	MICROCYTIC HYPOCHROMIC ADMIXED WITH MACROCYTES HYPERSEG NEUTROPHILS PLATELET ADEQUATE	NOT DONE	NOT DONE	-	NUTRITIONAL ANEMIA DUE TO IRON/B12/FOLATE
23	MILD MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA THROMBOCYTOSIS	NOT DONE	MASS WITH CENTRAL NECROSIS R LUNG,BRONCHIECTASIS	FNAC - MEDIASTINAL MASS - POSITIVE FOR MALIGNANCY SUGGESTIVE OF SQUAMOUS CELL CARCINOMA	ANEMIA OF CHRONIC DISEASE
24	MILD MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
25	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
26	MICROCYTIC HYPOCHROMIC RBCS WITH SEVERE ANISOPOIKILOCYTOSIS ADMIXED WITH NORMOCYTES	NOT DONE	NOT DONE	TFT- SUGGESTIVE OF HYPOTHYROIDISM	IRON DEFICIENCY ANEMIA
27	MICROCYTIC HYPOCHROMIC RBCS ADMIXED WITH NORMOCYTIC RBCS	NOT DONE	THALAMIC HEMORRHAGE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
28	NORMOCYTIC NORMOCHROMIC RBCS NEUTROPHILIA PLATELETS ADEQUATE	LUNG ABCESS	NOT DONE	BRONCHIAL WASH- INFLAMMATORY SMEAR	ANEMIA OF UNEXPLAINED ETIOLOGY
29	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
30	SEVERE MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA

31	NORMOCYTIC NORMOCHROMIC ANEMIA,NEUTROPHILIA AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
32	NORMOCYTIC NORMOCHROMIC RBCS LYMPHOCYTOSIS AND PLATELETS NORMAL	NOT AVAILABLE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
33	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
34	MODERATE MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	GRADE 3B BB FRACTURE R LEG	NOT DONE	-	IRON DEFICIENCY ANEMIA
35	DIMORPHIC ANEMIA LYMPHOCYTOSIS PLATELETS ADEQUATE	GARDEN TYPE III L SIDE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
36	DIMORPHIC ANEMIA WBCS AND PLATELETS NORMAL	R HIP PERIPROSTHETIC FRACTURE	NOT DONE	-	IRON DEFICIENCY ANEMIA
37	SEVERE MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS DECREASED	NOT DONE	LACUNAR INFARCT	-	IRON DEFICIENCY ANEMIA
38	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	ECG - POOR R WAVE PROGRESSION Q WAVES IN III, A VF	IRON DEFICIENCY ANEMIA
39	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
40	DIMORPHIC ANEMIA LYMPHOCYTOSIS PLATELETS ADEQUATE	OSTEOLYTIC LESION R SIDE OF JAW	NOT DONE	FNAC JAW LUMP- S/O OSTEOCLASTOMA	ANEMIA OF CHRONIC DISEASE
41	NORMOCYTIC NORMOCHROMIC RBCS LYMPHOCYTOSIS PLATELETS ADEQUATE	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
42	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
43	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
44	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT AVAILABLE	NOT DONE	UGI SCOPY- PYLORIC CANAL GROWTH - BIOPSY TAKEN,POORLY DIFFERENTIATED ADENOCARCINOMA	ANEMIA OF CHRONIC DISEASE
45	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
46	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	UGI SCOPY- ANTRAL GROWTH - BIOPSY - WELL DIFFERENTIATED ADENOCARCINOMA	ANEMIA OF CHRONIC DISEASE
47	MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
48	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
49	MICROCYTIC HYPOCHROMIC ANEMIA LEUCOPENIA PLATELETS ADEQUATE	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
50	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	INCREASED BV MARKINGS	NOT DONE	ECHO- CONCENTRIC LVH, EF 65 %,	ANEMIA OF CHRONIC DISEASE
51	MICROCYTIC HYPOCHROMIC ANEMIA LEUCOPENIA PLATELETS ADEQUATE	CARDIOMEGALY	NOT DONE	-	IRON DEFICIENCY ANEMIA
52	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA

53	MICROCYTIC HYPOCHROMIC ANEMIA LYMPHOCYTOSIS PLATELETS NORMAL	BILATERAL PLEURAL EFFUSION	NOT DONE	SPUTUM AFB POSITIVE	IRON DEFICIENCY ANEMIA
54	MICROCYTIC HYPOCHROMIC ANEMIA LEUCOPENIA PLATELETS ADEQUATE	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
55	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	INCREASED BV MARKINGS	NOT DONE	UGI SCOPY- GRADE II ESOPHAGEAL VARICES	IRON DEFICIENCY ANEMIA
56	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
57	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
58	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
59	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	BILATERAL PLEURAL EFFUSION	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
60	MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA AND PLATELETS NORMAL	INCREASED BV MARKINGS	NOT DONE	-	IRON DEFICIENCY ANEMIA
61	DIMORPHIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	BMA- ERYTHROID HYPERPLASIA WITH DIMORPHIC ERYTHROPOIESIS	IRON DEFICIENCY ANEMIA
62	MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA AND PLATELETS NORMAL	NOT DONE	NOT DONE	URINE CULTURE- E.COLI GROWTH	IRON DEFICIENCY ANEMIA
63	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT DONE	L SIDE INFARCT IN MCA TERRITORY	-	ANEMIA OF CHRONIC DISEASE
64	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA
65	MACROCYTIC RBCS HYPERSEGMENTED NEUTROPHILS PLATELETS ADEQUATE	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE/B12/FOLATE DEF
66	NORMOCYTIC NORMOCHROMIC ANEMIA NEUTROPHILIA PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
67	NORMOCYTIC NORMOCHROMIC RBCS WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
68	NORMOCYTIC NORMOCHROMIC ANEMIA NEUTROPHILIA PLATELETS NORMAL	NORMAL STUDY	NOT DONE	UGI SCOPY GROWTH LOWER ONE THIRD ESOPHAGUS BIOPSY SQUAMOUS CELL CARCINOMA	ANEMIA OF CHRONIC DISEASE
69	MILD MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
70	MILD MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	UGI SCOPY DUODENAL ULCER	ANEMIA OF CHRONIC DISEASE
71	NORMOCYTIC NORMOCHROMIC ANEMIA NEUTROPHILIA PLATELETS NORMAL	OLD CAVITY APICAL OPACITIES L LUNG	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
72	MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA AND PLATELETS NORMAL	MINIMAL BILATERAL PLEURAL EFFUSION	NOT DONE	-	IRON DEFICIENCY ANEMIA
73	NORMOCYTIC NORMOCHROMIC ANEMIA LYMPHOCYTOSIS PLATELETS NORMAL	BILATERAL PLEURAL EFFUSION	NOT DONE	SPUTUM AFB POSITIVE	ANEMIA OF CHRONIC DISEASE
74	MILD MICROCYTIC HYPOCHROMIC ANEMIA LYMPHOCYTOSIS PLATELETS NORMAL	BILATERAL PLEURAL EFFUSION	NOT DONE	-	IRON DEFICIENCY ANEMIA

75	MILD MICROCYTIC HYPOCHROMIC ANEMIA LEUCOCYTOSIS PLATELETS NORMAL	NORMAL STUDY	NOT DONE	URINE CULTURE- E.COLI GROWTH	ANEMIA OF UNEXPLAINED ETIOLOGY
76	MODERATE MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
77	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
78	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	ANEMIA OF CHRONIC RENAL DISEASE
79	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	L INTERTROCHANTERIC FRACTURE	NOT DONE	-	IRON DEFICIENCY ANEMIA
80	MODERATE MICROCYTIC HYPOCHROMIC ANEMIA WBC AND PLATELETS INCREASED	NORMAL STUDY	NOT DONE	UGI SCOPY ULCER IN PYLORUS	IRON DEFICIENCY ANEMIA
81	NORMOCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
82	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
83	SEVERE MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	LEFT THALAMIC HEMORRHAGE	-	IRON DEFICIENCY ANEMIA
84	NORMOCYTIC NORMOCHROMIC ANEMIA NEUTROPHILIA PLATELETS NORMAL	NOT DONE	MEDIASTINAL MASS	BRONCHOSCOPY- MASS RIGHT BRONCHUS,,BRUSH- SUGGESTIVE OF MALIGNANCY	ANEMIA OF CHRONIC DISEASE
85	MICROCYTIC HYPOCHROMIC ANEMIA NEUTROPHILIA AND PLATELETS NORMAL	BILATERAL PLEURAL EFFUSION	NOT DONE	-	IRON DEFICIENCY ANEMIA
86	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NORMAL STUDY	-	IRON DEFICIENCY ANEMIA
87	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
88	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NORMAL STUDY	-	IRON DEFICIENCY ANEMIA
89	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NORMAL STUDY	NOT DONE	-	IRON DEFICIENCY ANEMIA
90	NORMOCYTIC NORMOCHROMIC ANEMIA NEUTROPHILIA PLATELETS NORMAL	NORMAL STUDY	NOT DONE	FNAC INGUINAL NODE- CARCINOMATOUS METASTATIC DEPOSITS	ANEMIA OF CHRONIC DISEASE
91	MICROCYTIC HYPOCHROMIC ANEMIA BLASTS 23%, SEGMENTED 23% LYMPH 32% BASO 12% PL- NORMAL	NORMAL STUDY	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
92	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT AVAILABLE	NOT DONE	-	ANEMIA OF UNEXPLAINED ETIOLOGY
93	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NORMAL STUDY	PARAAORTIC NODES ENLARGED	-	ANEMIA OF CHRONIC DISEASE
94	NORMOCYTIC NORMOCHROMIC ANEMIA WBC AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	ANEMIA OF CHRONIC DISEASE
95	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	-	IRON DEFICIENCY ANEMIA

96	DIMORPHIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	NOT DONE	FNAC BREAST MASS- DUCTAL CARCINOMA GRADE III,,NODE-METASTATIC CARCINOMATOUS DEPOSITS	ANEMIA OF CHRONIC DISEASE
97	MICROCYTIC HYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT DONE	BRAIN NORMAL STUDY	-	IRON DEFICIENCY ANEMIA
98	MICROCYTIC NYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	NOT AVAILABLE	NOT DONE	-	IRON DEFICIENCY ANEMIA
99	MICROCYTIC NYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	BILATERAL BRONCHIECTASIS	NOT DONE	-	IRON DEFICIENCY ANEMIA
100	MICROCYTIC NYPOCHROMIC ANEMIA WBCS AND PLATELETS NORMAL	CARDIOMEGALY	NOT DONE	LEFT VENTRICULAR HYPERTROPHY	ANEMIA OF CHRONIC DISEASE